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Mutations and Design in Cellular Metabolism

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A. SUMMARY

This article is directed to the University undergraduate or biology teacher who is already familiar with cellular physiology. The main purpose of this paper is to demonstrate the unlikelihood of evolution philosophy which claims that mutations can be beneficial in some cases and that the long-term accumulation of beneficial mutations can advance an organism to a more complex form — for exampel, and eye-less trilobite to one which can see. It will be shown that mutations lead to both a deterioration of the organism and a reduction in the adaptive potential of the species to future environmental changes. This would be more compatible with a creation philosophy of initally created complex life forms subsequently affected by deteriorative processes.

Thus the article surveys what mutations are, and their effects on cellular metabolism. Natural selection is defined and its effect on mutational change in living organisms is briefly described. Other factors relating to the effects of mutations on cellular metabolism are also discussed.

B. INTRODUCTION

1. The Evidence for Order in Living Cells

It is common knowledge that the basic unit of life, the self-replicating cell (Figure 1), can be likened to a highly co-ordinated and efficient assembly plant or factory. The compartmentalized nature of biochemical reactions within the cell, and their interactions with events occurring in other regions within the same cell, is one of the highlights of the modern approach to the study of cell physiology. Thus the nucleus, containing the DNA or genetic

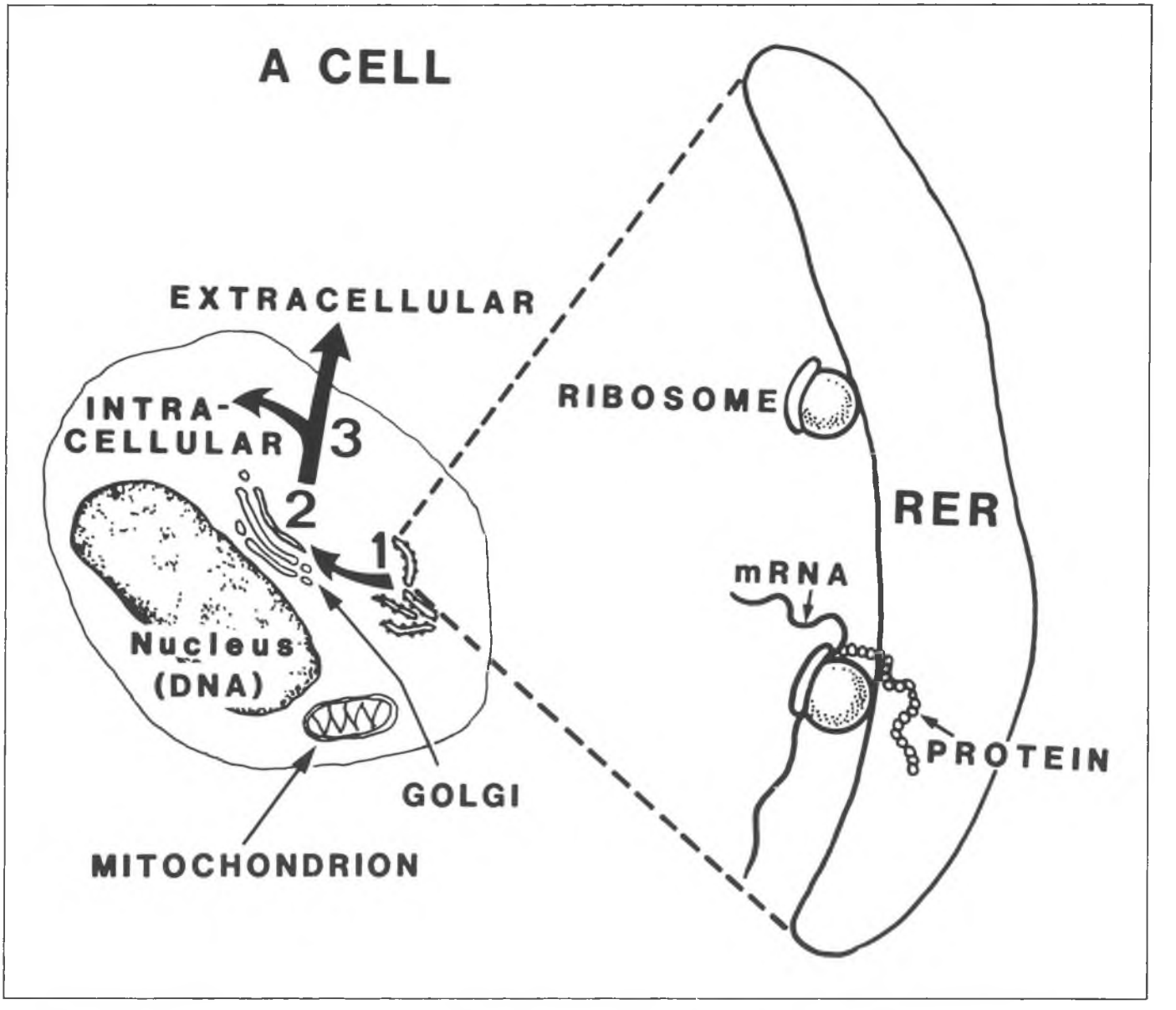

Figure 1. *The living cell is organized similar to a factory. Information in the form of messenger RNA is sent from the nucleus to be translated, into protein, by the ribosome. The ribosome is attached to the rough endoplasmic reticulum (RER). Newly synthesized proteins are transferred from the internal space of the RER (1) to the golgi apparatus (2). Proteins are then transferred to internal or extracellular destinations (3). The mitochondria are the power plants of the cell.*

content of the cell, can be considered to be similar to the director's office where the blueprints for products to be made are kept, along with the office records. When messenger RNA is transcribed from the DNA and is transferred from the nucleus to the ribosomes (attached to the rough endoplasmic reticulum (RER) in the cytoplasm), the analogy corresponds to the foreman taking a particular plan from the office into the factory production area and handing it to the machine operator. The ribosome, or machine operator, then translates the RNA, or plan, into a specific product. In the cell this product is one of 50,000–100,000 specific (different types of) proteins.[1] Each protein has a very specific structure/function relationship (Figure 2). The power to operate the cell comes primarily from the mitochondria (or in plants, the mitochondria and chloroplasts). A factory would similarly be supplied with energy from a nearby power plant.

Thus, the cell can be compared to an ordered factory in many ways. From this perspective it could be argued that just as intelligence was needed to assemble and develop the factory and its products, so intelligence, that is, a Designer, was needed to put together the living cell as a complete unit. In fact, biology textbooks often use phrases such as "the

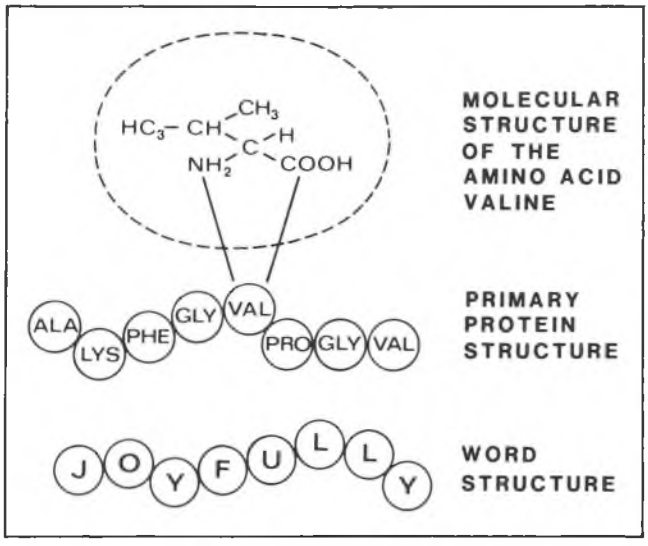

Figure 2. *Proteins (and DNA) have a high degree of information content, somewhat analogous to word structure. Just as random letter insertions or deletions scramble the meaning of a word, so mutations that result in an amino acid change alter the structure, and usually the functional capability of that protein.*

intricate design of the living cell. . ." already unconsciously admitting this truth.

Evolutionists conclude, however, that deliberate design is not evident in the cell. Theodore Dobzhansky, a well known evolutionary biologist, has stated, "theologians of the nineteenth century erroneously claimed that the directive organization of living beings evinces the existence of a Designer."[2] He and others believe that complex life has evolved by a series of 'mutations' acted upon by 'natural selection'. Ernst Mayr has stated, "It must not be forgotten that mutation is the ultimate source of all genetic variation found in natural populations and the only new material available for natural selection to work on."[3]

2. What are Mutations?

Gardner has defined mutations as "a change in the DNA at a particular locus in an organism."[4] However, a more complete definition would be: "a **random** breakage/recombination or insertion event that alters the previous function of the DNA coding region".

The word 'random' is necessary in the definition because there are some recombinational events in DNA which are ordered, and functionally purposeful. For example, during thymocyte maturation, recombination of the DNA genes coding for antibody formation occurs in a precise manner so that the protein product presents a recognition site for specific antigens.

3. Can Mutations Followed by Natural Selection Account for the Ordered Complexity of Life? Two Models.

Henry Morris has stated, "There are basically only two possible models of earth history. . .In the evolution model, the entire universe is considered to have evolved by natural processes into its present state of high organisation and complexity. . .The creation model, on the other hand, defines a period of special creation in the beginning, during which the basic systems of nature were brought into existence in completed, functioning form right from the start."[5] There are several corollaries to each. Evolution is assumed to have occurred only under presently known laws of nature (uniformitarianism); that life forms have evolved from simple to complex, and it requires long ages. Creation implies that living organisms were complex to begin with, but may now be in a process of deterioration. Therefore the laws of nature must have been transcended in that initial creation period by the Creator, and so this period could have occurred relatively recently.

Within the evolutionary framework, mutations are viewed as the rearrangement of DNA causing the appearance of new genetic material upon which natural selection acts to favour the survival of new and, generally speaking, more complex living things. Within the creation framework, mutations are considered to be a degenerative action upon a highly organized information code (the genetic code), resulting in deformed organisms, many of which would subsequently be removed by natural selection. But not all would necessarily be removed, and therefore it would be expected that some defects would be passed from generation to generation resulting in the overall increasing deterioration of living things with the passage of time (for example, colour blindness in humans).

The observations of science concerning mutations can be 'fitted' within either framework (Figure 3). No one has yet observed a fish evolve into a reptile, etc. Neither has anyone observed a fish created 'ex nihilo' (except God). In fact, observing the effects of mutations can be likened to watching only one or two picture frames from an entire motion picture film. Therefore, it can be stated that empirical science can **not** definitively answer questions about the past or even the future. But empirical science can answer two basic kinds of questions:

(a) classification — height, weight, composition, etc.; and

(b) change within the present — where the

observer is able to measure both precursors and products.

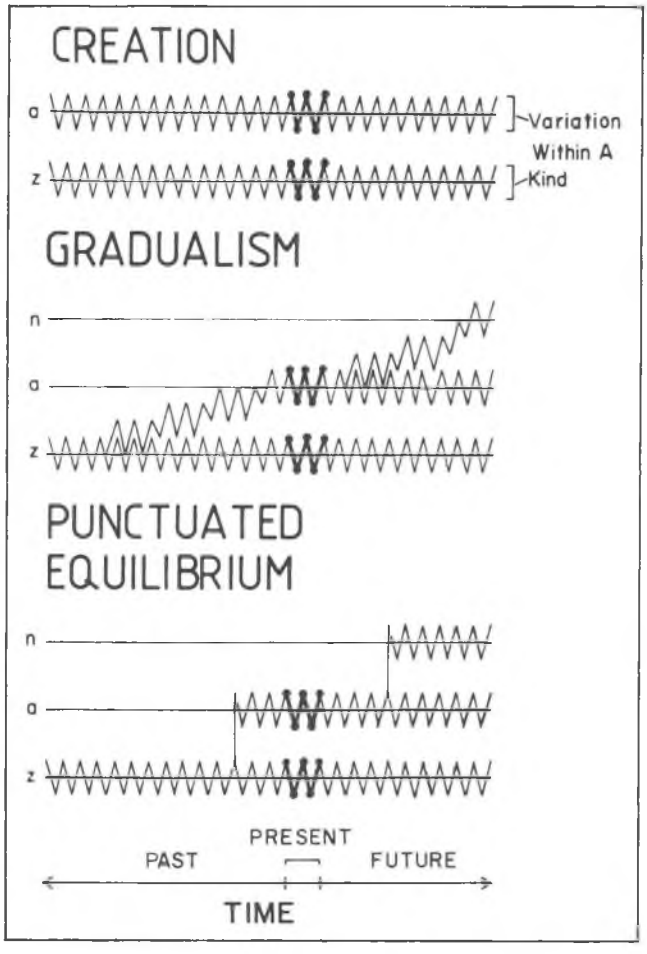

Figure 3. *There are two major philosophical frameworks in which to place the observations of science, Creation or Evolution. Gradualism (Darwinism) and Punctuated Equilibrium are the two present popular mechanisms of evolution. (a) and (z) are two "kinds" of organisms, unrelated reproductively and morphologically. (n) indicates a "theoretical" newly evolved organism. The zigzag line indicates the variation observed in different groups of organism (for example — dogs, cats).*

From the observations of empirical science, extrapolations can be made concerning the past or future, but these are heavily influenced by philosophical bias. Even though this is the case, the question can be asked: which model do the observations on the effects of mutations support with the least number of secondary assumptions? The model with the least number of secondary assumptions is more likely the correct one. It will be contested in this article that the overall effect of mutations results in the loss of information content (degradation) and that this is more readily supportive of the creationist philosophy than the evolutionary philosophy.

C. EXAMPLES OF EFFECTS AND COROLLARIES OF MUTATIONS

There are several groups of observations or data that need to be considered when evaluating the effect of mutations upon living animals or plants. These are:

1. General effects of mutations;
2. Design/optimized structure;
3. Probability;
4. Natural selection;
5. Gene load;
6. Speciation/classification; and
7. Fossil record.

1. General Effects of Mutations

One of the first things that should be noted is that mutations have been one of the prime tools used in investigating cellular metabolism. For example, if certain selective media are used, only mutant organisms will grow in that medium and a build-up of precursors to the defective step in metabolism will occur. By isolating different mutants, the precursor steps leading up to the defective enzyme(s) can be determined, and eventually an overlap in information can occur such that a biosynthetic pathway is determined. Lehninger has commented, "Such mutant micro-organisms, in which one enzyme or another is defective, are powerful tools for study of metabolism."[6] Thus, in these cases, mutations are very helpful in investigative work, but are hardly helpful for the cell with the defective enzyme. As C. Satoh has stated concerning mutations in general, "more recently, there is direct evidence from *Drosophila* that the majority of spontaneous and induced mutations are accompanied by loss or inactivity of gene product, and there is similar evidence from the mouse regarding induced mutations."[7] Theoretically, every one of the 50,000–100,000 proteins in a cell could be initially mutated, with varying resultant effects on cellular metabolism.

Listed below are brief descriptions of only a few isolated examples of known protein mutants in humans and their effects on the five basic cellular metabolic pathways (that is, protein, amino acids, carbohydrate, lipid, and nucleic acid).

(a) Disorders of protein (collagen) metabolism (Figure 4; Table 1)

Collagen is a good example of a protein in which to study the effects of mutations. It is a large protein with molecular weight of 100,000–150,000 daltons

Table 1: Defects Associated With Collagen Disorders

Disorder	Types	Clinical Manifestations
Ehlers-Danlos Syndrome (EDS)	8	hyperextensibility of joints, skin bruising and fragility
Osteogenesis Imperfecta (OI)	4	fragile bones and bone deformities, blue sclerae, dental abnormalities, sometimes deafness
Marfans Syndrome (MS)	4	arterial degeneration, long extremities, lens displacement
Epidermolysis Bullosa (EB)	5	blister formation with skin lesions
Cutis Laxa (CL)	4	laxity of skin, systemic complications

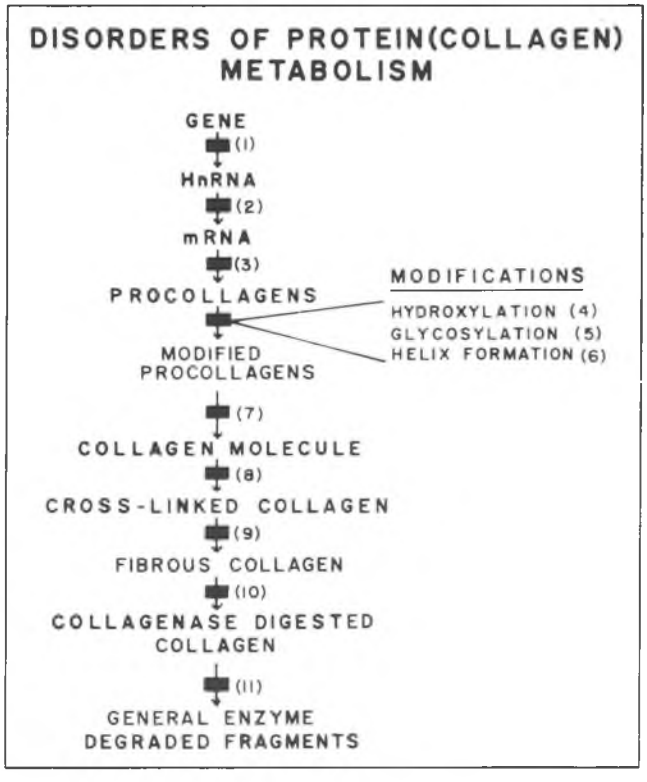

Figure 4. *Disorders of human protein (collagen) metabolism. Observed locations in enzyme deficiencies (due to a mutation) metabolising given steps are indicated by ■. Patients have been identified that are affected by blockages at each one of these sites. In most cases the lack of the product beyond the block, or an excess of precursor before it, or an improperly processed product, usually results in a disease condition (see text).*

per monomer chain.[8] Three chains, in the correct proportion, are needed to make up a helical molecule which is processed, modified, and transported extracellularly before being cross-linked into a macromolecular fibre. The mRNA for the ∝2 chain is transcribed from DNA containing 52 introns.[9] Needless to say it is a very complex process, and as can be seen from Figure 4, there are known multiple defects that can occur in humans at every step of the process. Some of these defects which are known to have occurred at each 'blocked' step are listed (the numbers correspond to those given in Figure 4 and abbreviations with those in Table 1):[10-13]

(1) Possible lack of gene transcription;

i. No type III collagen results in EDS IV,

ii. Deficient pro∝2 (I) chains production results in OI II,

iii. No pro∝2 (I) chains results in OI III, IV.

(2) Incorrect removal of introns or exon ligation may result in some forms of OI.

(3) Excess collagen production, possibly due to excessive mRNA may result in fibrosis. Decreased collagen production may result in EDS IV (no type III collagen), OI II [decreased pro ∝2 (I)], or OI III, IV [no pro∝2 (I)].

(4) i. Deficiency of prolyl hydroxylase results in decreased triple helix stability. This can cause scurvy, tissue anoxia, and decreased secretion of collagen.

ii. Deficiency of lysyl hydroxylase results in decreased glycosylation and/or cross-linking, and can cause EDS IV, alkaptonuria, some forms of OI, and scurvy.

(5) Deficiency of hydroxylysyl glycosylase affects cross-links formation and fibre diameter, and this can result in dominant EB.

(6) i. Structural mutation (pro∝2^{cx}) in the c-propeptide prevents trimer association, and results in OI.

ii. Structural mutants (pro∝1^{s} , pro∝2^{l} , and pro∝2^{s}) shorten (S) or lengthen (L) the pro∝ chains and prevent helix formation. This results

in protein suicide (excess collagen degradation intracellular), and also manifests as: pro∝1^{s} (OI), pro∝2^{L} (MS), pro∝2^{s} (OI or OI-EDS).

(7) i. Lack of removal of propeptides results in incomplete cross-linking within the triple helical collagen molecule, and this results in dermatosparaxis, EDS VII.

ii. Structural mutant (pro∝2) prevents cleavage of the amino terminal propeptide by procollagen N-proteinase, and results in EDS VIII.

(8) Deficiency of lysyl oxidase results in EDS, CL.

(9) Fibrillogenesis defects result in EDS I-III.

(10) Altered rates of degradation of collagen by collagenase occur in rheumatoid arthritis, recessive dystrophic EB, Paget's disease of the bones, hyperparathyroidism, hyperthyroidism, tumour invasion, uremia, and inflammatory processes involving leukocyte collagenase.

(11) Altered rates of removal of degradation products (of collagen) occur in hydroxyprolinemia and hydroxylysinemia.

(b) Disorders of Amino Acid Metabolism (Figure 5)

The defects associated with phenylalanine and tyrosine are only representative of a host of disorders associated with amino acid metabolism.[14–16] Some of these 'blocked' steps are:

(1) A deficiency of phenylalanine hydroxylase results in phenylketonuria. "Children with this disease are severely mentally retarded, frequently have convulsions, and often have to be institutionalized."[17]

(2) A deficiency of this enzyme results in congenital thyroxine deficiency (cretinism). Cretinism is "characterized by mental retardation, slow body development, dwarfism, and a characteristic facial structure."[18]

(3) A deficiency of tyrosine 3-mono-oxygenase results in albinism. This results in a "Lack of melanin pigment formation in the skin, hair, retina, and choroid coat of the eye. . .such individuals are called albinos."[19]

(4) A deficiency of tyrosine-glutamate aminotransferase results in the rare disease tyrosinosis. Pronounced increase of tyrosine and hydroxyphenylpyruvate are formed in the plasma. "In one case recently described, an infant male, multiple congenital anomalies including microcephaly was reported."[20]

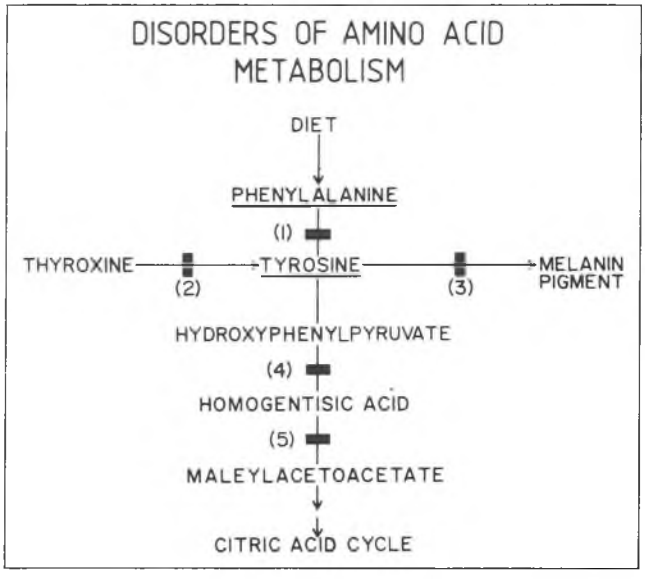

Figure 5. *Disorders of human amino acid metabolism. See Figure 4 for legend.*

(5) A deficiency of homogentisic acid oxidase results in a condition called alkaptonuria. Homogentisic acid builds up and is excreted in the urine. The urine may be dark coloured because of the oxidized acid. "The condition is usually benign, although there may be pigmentation of cartilage and other connective tissues in later life."[21]

At least 43 inborn errors of amino acid metabolism have been identified in humans.[22]

(c) Disorders of Carbohydrate Metabolism (Figure 6)

Again, only a small representative portion of carbohydrate metabolic disorders are listed.[23–24] Some of the 'blocked' steps are:

(1) A deficiency of galactokinase leads to excess galactose production, that is, galactosemia, and cataract formation.

(2) A deficiency of UDP-glucose: D-galactose 1-phosphate uridylyltransferase results in galactosemia as well. "As a result D-galactose and D-galactose 1-phosphate cannot be metabolized and accumulates in the blood and tissues. The liver and other organs become enlarged, vision becomes impaired because of the formation of cataracts, and there is mental retardation."[25]

(3) A deficiency of UDP-glucose-4-epimerase (NAD) also results in a galactosemia.

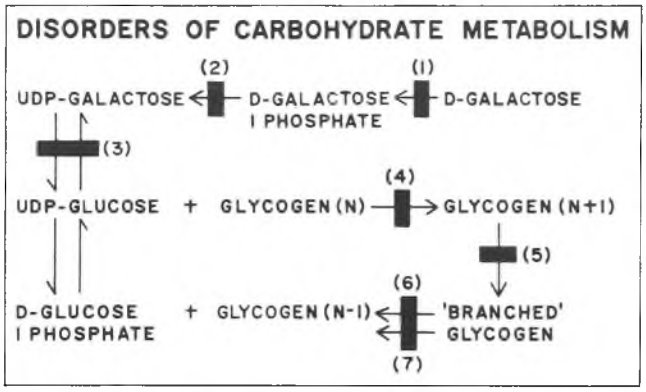

Figure 6. *Disorders of human carbohydrate metabolism. See Figure 4 for legend.*

(4) A deficiency of glycogen synthase results in the glycogen storage disease type 9.
(5) A deficiency of glycosyl-4-transferase (or transglycosylase) results in the glycogen storage disease type 4 (Anderson's disease).
(6) A deficiency of amylo-1,6-glucosidase results in the glycogen storage disease type 3 (Cori's disease).
(7) A deficiency of glycogen phosphorylase results in the glycogen storage disease type 5 (McArdle's disease in muscle) and type 6 (Hers' disease in liver).

At least 40–50 diseases have been identified in humans that are related to molecular defects in the metabolism of carbohydrate.[26] Most are severe disorders.

(d) Disorders of Adrenal Steroid Biosynthesis (Figure 7)[27]

Some of the 'blocked' steps are:

(1) A deficiency of the enzyme desmolase is incompatible with life, since no steroid hormones can be produced.
(2) A deficiency of 3β — hydroxydehydrogenase is also incompatible with life, and no affected infants have lived more than a few months.
(3) A deficiency of C-17 hydroxylase results in the accumulation of excess 11-deoxycorticosterone, and a hyperaldosterone-like syndrome develops. "Since the C-17 defect is present in the ovaries and testes, a deficiency of both male and female hormones occur and both sexes are of the female phenotype, regardless of genetic sex."[28]
(4) A deficiency of C-21 hydroxylase inhibits both glucocorticoid and mineralocorticoid pathways.

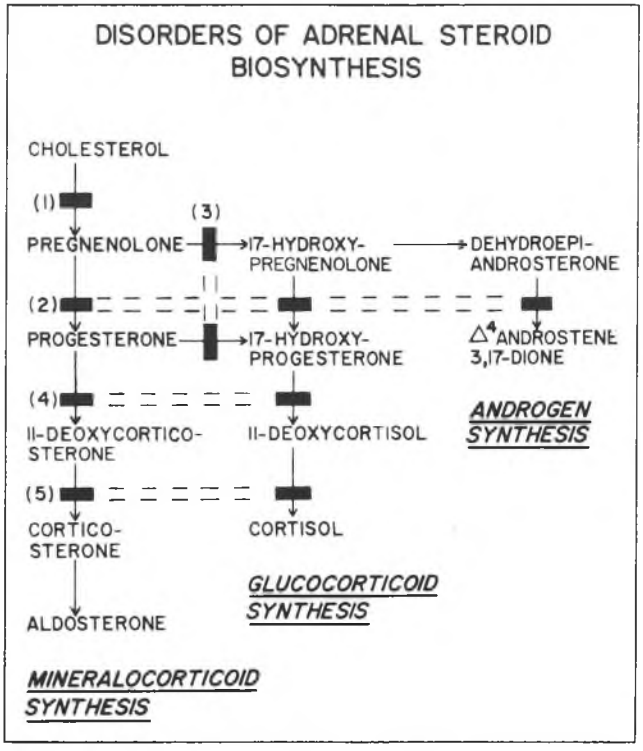

Figure 7. *Disorders of human adrenal steroid biosynthesis. See Figure 4 for legend. (Reprinted with permission from the 'Adrenal Cortex', published by the Upjohn Co., Kalamazoo, 1975, p. 63.)*

This leads to a spillage of intermediates into alternate pathways, the result being an increased production of testosterone in the peripheral tissues. "*In utero* and at birth, varying degrees of virilization in the female are found. . .in its mildest form, the androgenic effects may only be manifest at puberty, for example, hirsutism, poor breast development and amenorrhoea."[29]
(5) A deficiency of C-11-B-hydroxylase results in a condition similar to the C-21 hydroxylase defect. However, deoxycorticosterone, a potent mineralocorticoid, is formed in excess and produces hyperaldosteronism as well as virilization. "This syndrome presents early virilization with hypertension and hypokalemia."[30]

J.M. Orten in another review lists 23 known human enzyme defects in lipid metabolism.[31]

(e) Disorders of Nucleotide and Nucleic Acid Metabolism (Figure 8)[32]

Some of the 'blocked' steps are:

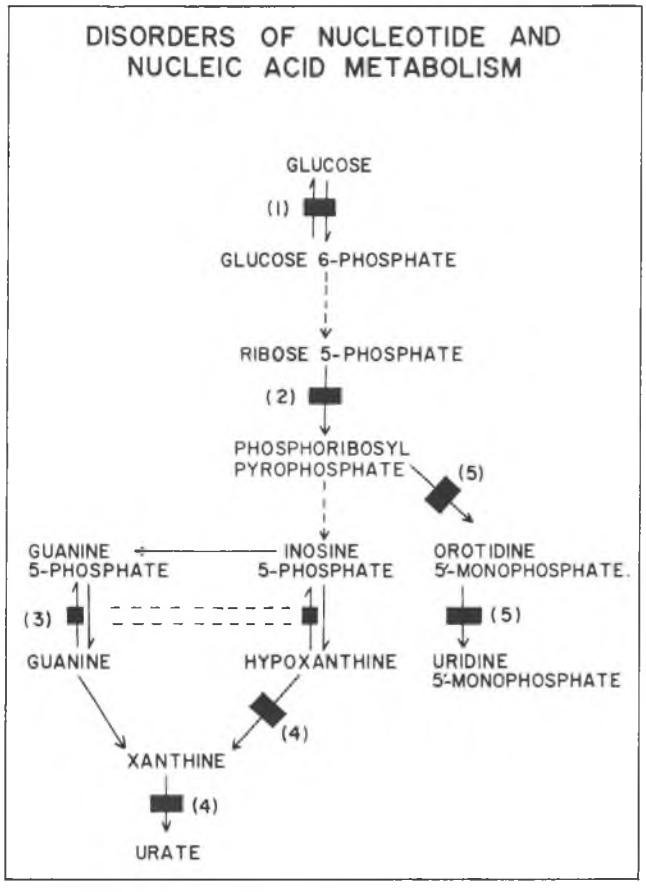

Figure 8. *Disorders of human nucleotide and nucleic acid metabolism. See Figure 4 for legend.*

(1) A deficiency of glucose-6-phosphatase allows for increased urate production, and this may result in gout, "a very painful arthritis caused by the crystallization of monosodium urate in a synovial cavity. . ."[33] Long-standing hyperuricaemia causes deposition of urate in the kidneys and chronic renal impairment (urate nephropathy).

(2) A deficiency of 5-phosphoribosyl 1-pyrophosphate (PRPP) synthetase results in increased activity due to a defect in the ability of the enzyme to 'switch-off' in response to high nucleotide levels. This can lead to the condition of gout due to excessive purine synthesis.

(3) A partial deficiency of hypoxanthine-guanine phosphoribosyl transferase (HGPRT) results in defects in the salvage pathway and can result in gout. A complete deficiency causes the inborn error of metabolism, the Lesch-Nyhan Syndrome, "Patients appear normal for the first few months of life but then growth slows and neurological process is delayed. After the age of two the full syndrome is apparent with spasticity, involuntary movements, mental retardation, gout, uric acid calculi and a compulsion for self-mutilation."[34] This complete deficiency of HGPRT can be caused by several different types of mutation: duplication of exon 2, 3, a deletion, or a point mutation.[35]

(4) Increased activity of xanthine oxidase may result in gout.

(5) A deficiency of orotate phosphoribosyl transferase (OPRT) / orotidine - 5 - phosphate decarboxylase, a protein with two enzyme activites, results in the disorder of pyrimidine synthesis — hereditary orotic aciduria. Symptoms are as follows, "Patients are normal at birth but become severely anaemic during the first six months of life. . .some have a degree of mental retardation."[36]

It must be concluded from this brief overview of mutations affecting protein, amino acids, carbohydrate, lipid and nucleic acid metabolism that mutations are harmful. In fact, it could easily be argued that out of the several thousand known human metabolic defects not one has been shown to really be beneficial to the individual person, much less substantial enough to be supportive of the concept of macroevolution of a human from another type of animal, for example, the chimpanzee.

It should also be noted that of 1,876 genetic disorders listed by McKusick, 783 were autosomal recessive. This becomes important when considering gene mutational load (see below).[37]

An analogy of the effect of mutations on cellular metabolism could be likened to a man with a chisel and hammer randomly knocking out one or two teeth from any one of the gears in an old grandfather clock. The clock would still run, but would 'clunk' periodically. This is like a point mutation in the DNA which results in one altered amino acid in the protein. If he then knocked whole gear out of the clock it might still run, but now the second hand may be non-functional. This would be like a deletion event during recombination which would result in several altered amino acids. Finally, if he chopped the counter weights off, the clock would stop. This would be analogous to any lethal mutation, especially non-disjunction or translocation events which result in certain proteins completely lacking. Or another way of looking at the effects of mutations on the genetic information content of a cell (which determines its metabolic characteristics) is to compare it to the information content in the written letters and words of a book. If one page were taken and cut in half longitudinally (perpendicular to the writing), and then shifted one line out of order, the page could still be read but some words would be meaningless. This could be likened to a point mutation. If another slice was made and the pieces shifted or even one of them

removed this would be similar to frameshift and deletion mutations. This could be repeated again and again. Each successive change would render the information content of the page more meaningless. So it is with mutations and their effects on cellular metabolism.

As W.R. Thompson has stated concerning mutations, "If we say that they are useful, we are still speaking too leniently. In general, they are useless, detrimental or lethal."[38] The very existence of complex and regulated biochemical pathways, unmutilated in healthy persons, speaks clearly of deliberate design (Romans 1:20).

2. Design and Optimization

R.C. Lewontin, in discussing adaptations stated, "It was the marvellous fit of organisms to the environment, much more than the great diversity of forms, that was the chief evidence of a Supreme Designer. Darwin realized that if a naturalistic theory of evolution was to be successful, it would have to explain the apparent perfection of organisms and not simply their variation."[39]

Can evolutionary adaptation not only account for organisms 'adjusting' to new environments, but actually 'mutating' into totally new organisms adapted to new environments, for example, amphibian to reptile? Or, are there constraints suggested by design and optimization features that argue against this? Does design and optimization support the concept of an intelligent Designer?

(a) Design

There are several levels of organization in which to observe design in living things. These are: the molecular, organ (tissue), whole animal, morphogenesis, and habit or intelligence levels.

(1) At the molecular level the enzyme ATP-synthetase will be discussed (Figure 9).[40–41] It converts low energy potential ADP to high energy potential ATP when protons are forced through a special channel. There are eight protein subunits that are part of this high molecular weight enzyme (450,000 daltons). Mutations in any one of these proteins can destroy the function of the entire unit. All the ATP-synthetase units in a cell must be assembled on the correct membranes, in correct orientation so as to not short circuit the membrane. In addition, each protein subunit must be added in the correct order during assembly otherwise the membrane will leak.

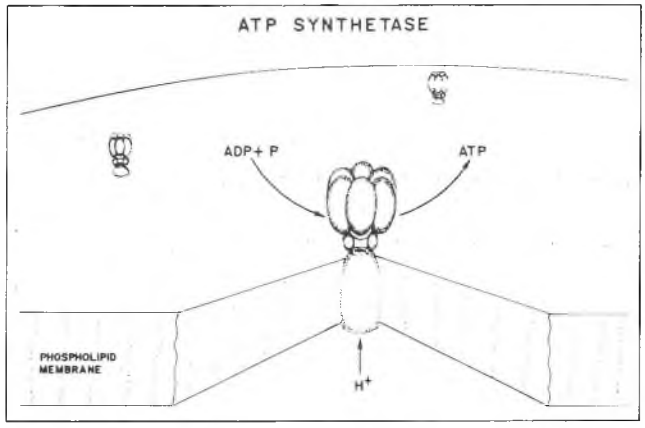

Figure 9. *Evidence of design at the biomolecular level — ATP-synthetase. ADP (adenosine diphosphate) is converted to ATP (adenosine triphosphate) by the ATP-synthetase. Hydrogen ions (H+) are utilized in this conversion. This enzyme is a cluster of eight proteins, and is associated with certain phospholipid membranes within the cell.*

There is a threshold potential value that associated proton pumps must obtain, or there will be no net synthesis of ATP — it has an all or nothing functional capacity. Organisms from bacteria to man have this complex enzyme.
The ATP-synthetase pump reveals design. It only functions as a complete complex unit, and this unit is found in all forms of life. Mutations are destructive to its function.[42]

(2) At the level of organ function the mammalian aorta is a good example of design (Figure 10). In man, the blood is pumped out of the left ventricle in large surges. The vessel into which it is pumped must accommodate these surges. The aorta does just that. It is more elastic proximal

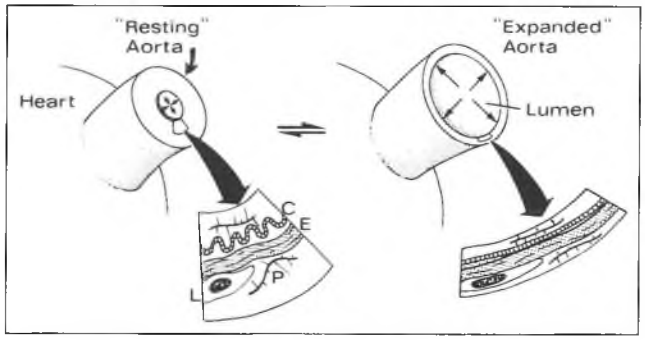

Figure 10. *Evidence of design at the organ level — the aorta. This circulatory vessel is composed primarily of the proteins elastin (E), collagen (C), and proteoglycans (P). Cells are labelled as (L). The aorta expands during a pulse of blood from the heart, but immediately after contracts to a "resting" position.*

(near) to the heart and decreases in elasticity distal (away) from it. This is due to the type and quantity of macromolecules present in the aortic

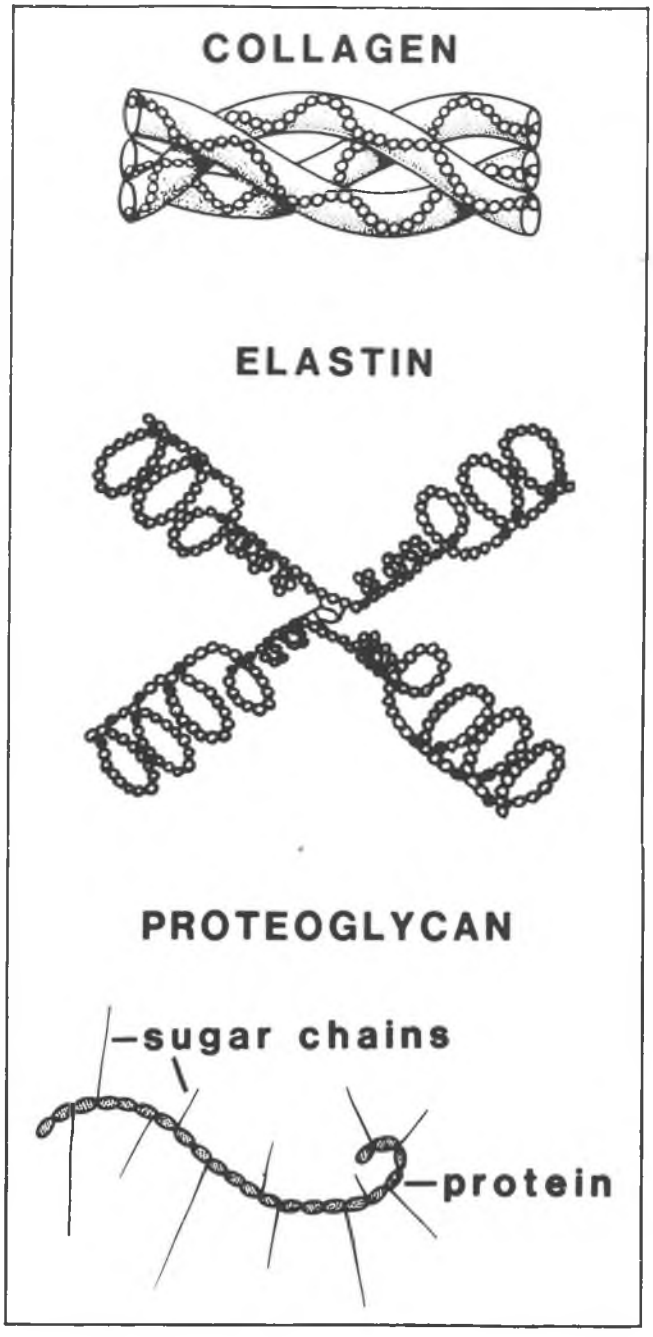

Figure 11. A diagram of the three major protein components of the aortic wall.

wall. The predominant ones are the proteins collagen, elastin, and proteoglycans (Figure 11).[43] Collagen is a helical molecule which is cross-linked into large fibrous strands (much like a hemp rope). It is very flexible, but also tensile. In fact, per unit dry weight it is equivalent to the strength of steel. When the aortic wall is contracted (no blood surge), the collagen is folded into bundles. As a new surge of blood expands the aortic wall, the collagen unfolds until it is linear. Once this length is reached it exerts maximum resistance and will stretch only 2% further before damage is done.[44] Thus, the collagen limits the expansion of the arterial wall. Elastin's function is what its name implies — elasticity. Elastin in the contracted aorta is an amorphous mass. Upon extension of the arterial wall, elastin expands, up to 200% of its resting length.[45] It therefore does not limit arterial wall expansion as much as collagen. But it is responsible for wall retraction once the lumen blood pressure has dropped.

In several disease states like Marfan's Syndrome, which affects collagen[46] and elastin[47] structure or composition in the aorta, aortic ruptures (or aneurysms) frequently occur.

It is important to note that the ratio of elastin to collagen is 2:1 in the region near the heart and reverses around the level of the diaphragm (Figure 12).[48] This accounts for the increased elasticity near the heart. If the opposite occurred, the aorta would most likely burst.

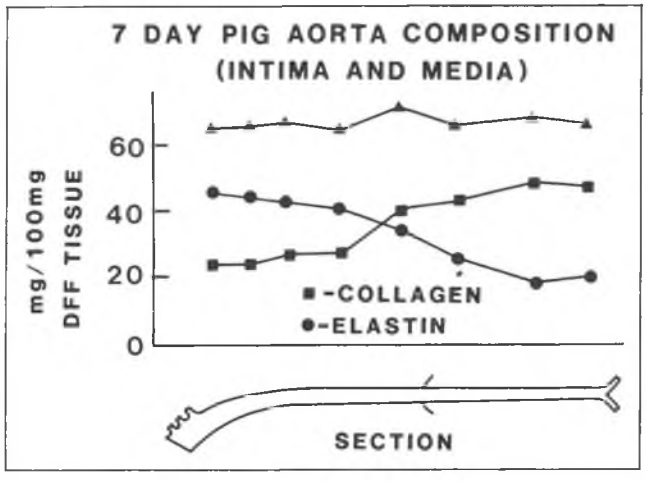

Figure 12. The absolute amounts of elastin to collagen in different anatomical sections of the seven day old pig aorta. ● Elastin, ■ Collagen, ▲ Total Protein, DFF — Dried Fat Free weight.

There are also several different types of collagen in humans, and the ratio of abundance of some of these appears to be important for normal aortic wall function.[49] As was noted earlier in the effects of mutations on protein metabolism, an imbalance of synthesis of various collagen molecules is usually deleterious.

Proteoglycans are important too.[50] They act as filters to soluble molecules because of the high polyanionic charge of the sulphate groups on the polysaccharide chains. They may also act as recoil bumpers. As the elastin contracts the aortic wall, the proteoglycans become enfolded. As the polyanionic groups come close together a repulsion is set up, and thus contraction is

Table 2: Mean ± Standard Deviation of Hydrophobic Index, % Elastin, Blood Pressure, and Heartbeat in the Aorta

Species	(n)	Hydrophobic Index[a]	(n)	% Elastin[a]	(n)	Average BP[b]	(n)	Average Heartbeat[b]
Birds	(3)	33.2 ± 2.1	(3)	45.7 ± 7.4	(11)	151.0 ± 31.0	(4)	109.5 ± 50.7 Low[d] 396.0 ± 103.2 High
Mammals	(5)	23.2 ± 1.9	(6)	59.8 ± 16.4	(17)	104.1 ± 32.7	(9)	102.0 ± 52.4 Low[d] 370.3 ± 166.0 High
Reptiles	(1)	23.7	(1)	33.0	(2)	57.5 ± 44.5	(2)	33.3 ± 1.8
Amphibia	(2)	15.7 ± 0.8	(2)	45.0 ± 26.9	(3)	33.0 ± 12.4	(2)	40.0 ± 7.1
Fish	(20)	10.2 ± 2.9	(17)	23.9 ± 11.4	(8)	44.8 ± 21.5	(8)	47.9 ± 16.6
Crustaceans		0[c]	(3)	0[c]	(3)	7.8 ± 0.8	(1)	45
Gastropods		0[c]	(1)	0[c]	(1)	30.0	(1)	28

[a] From H. Sage and W. Gray, Comp. Biochem. Physiol. 68B, 473-480 (1981).

[b] From P.L. Altman and D.S. Dittmer, Biology Data Book, 239-241 (1967).

[c] From the observations of Sage and Gray (ibid), no elastin content is found in the invertebrates, therefore both HI and % Elastin are by definition 0.

[d] Because of the great dispersity in mammalian and bird heartbeat rates (46-738), these groups were divided into Low (< 200) and High (> 200).

slower or stopped. The cartilage-deficient mouse[51] is a good example of what happens when little or no proteoglycan is present due to mutation. The result is that the cartilage collapses and does not retain its shock resistant properties.

It is clear that the composition of the aorta corresponds directly to the blood pressure of the organism, and indirectly with the heartbeat (Table 2, Figure 13). This is a beautiful example of a structure/function relationship, but does not necessarily imply evolution from a common ancestor. If it did then in using these criteria birds would more likely have evolved from mammals (not reptiles as some suppose).

These three macromolecules, among many others, work synergistically together. There are many known human disorders due to mutations that affect the function of these proteins and thus the arterial wall (and any other connective tissue), but none has been shown to improve the design or function of it.

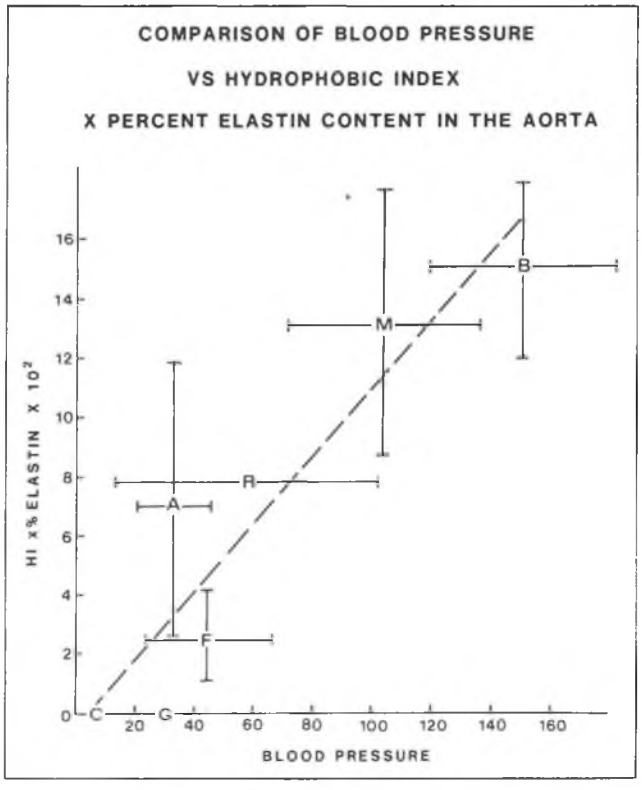

Figure 13. *A graph of blood pressure (BP) versus percent elastin times the hydrophobic index (HI). There is a clear correlation (using a least squares regression analysis, r = 0.91) between the BP and the percent elastin times the HI. The hydrophobic index is a measure of the hydrophobicity of the animo acids within the elastin from each organism. Birds (B), Mammals (M), Reptiles (R), Amphibians (A), Fish (F), Crustaceans (C) and Gastropods (G). Calculated from data in Table 2. Values are the mean ± one standard deviation.*

(3) At the level of body function the bombardier beetle is a good example of design (Figure 14).[52-53] This beetle reveals a co-operation among various organs within its body that defies explaining its presence by evolutionary selection of mutations. It has a defensive mechanism which involves 'twin guns' on the rear of its abdomen. These guns fire water and quinones (offensive to many animals), at 100°C and with O_2 which causes a pop to be heard. The firing of either gun requires the co-ordinated action of two glands, the reservoir, and the vestibule. Hydroquinones and hydrogen peroxide are secreted by gland cells into the reservoir lumen, and stored there. A

sphincter muscle separates that gland from the vestibule. Catalase and peroxidase are secreted into the vestibule at the appropriate time, and the sphincter muscle releases the contents of the reservoir into the vestibule. When this occurs, an explosive reaction results converting the hydroquinones to quinones, and hydrogen peroxide to oxygen and water at 100°C. This is 'shot' out the tail gun in virtually any direction the beetle desires, usually in response to an aggressor.

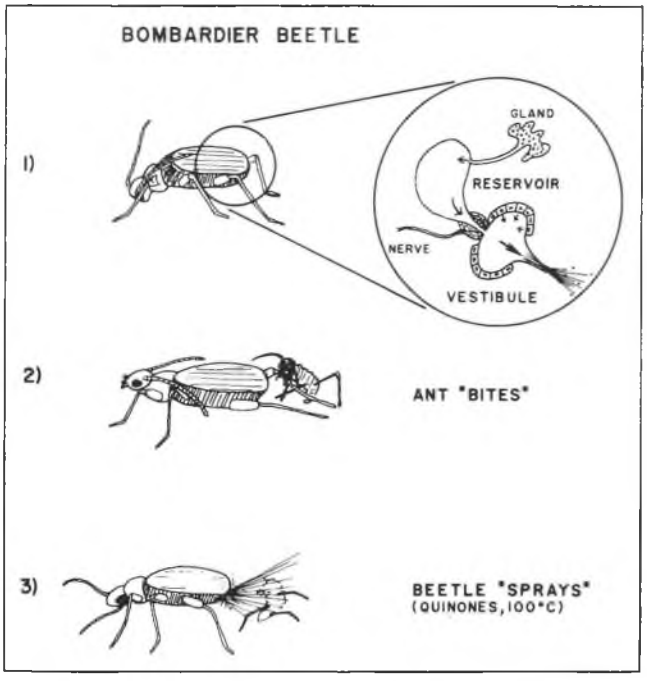

Figure 14. *Design at the level of body organization — the "cannon" in the bombardier beetle. The secretory contents of the reservoir gland must be kept separate from those delivered into the vestibule, until they are used. Once they are mixed, a rapid explosion occurs which results in the release of quinones, O_2, H_2O at 100°C. These are spewed out of an orifice at the rear of the beetle's abdomen. It is often used as a defensive weapon.*

Could mutations have been used to 'design' these organs? The secretory cells for the correct reagents have to be segregated into separate glands or else it would be self-destructive. A control muscle between the two glands is essential (the sphincter). An outlet to release the gases and hot liquid is necessary just as is a nervous system for control. The entire structure must be functional, anything less would be deleterious to the beetle.

(4) Design in development is revealed in the morphogenetic change that occurs when a tadpole develops into a frog (Figure 15).[54] It is a dramatic example of a well orchestrated and regulated increase in expression of some

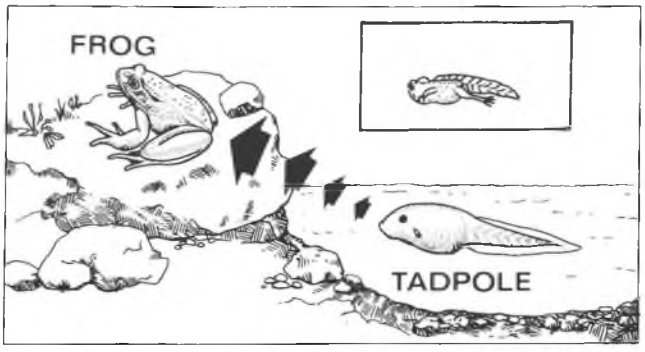

Figure 15. *Design at the level of development — the tadpole to frog transition. This morphogenetic event requires precise, coordinated, concerted, and timed expression of various enzymes. If thyroxin is artificially added, the tadpole prematurely develops arms and legs (insert).*

enzymes and decrease of others. Certainly one of the main enzymes involved is collagenase. Its expression is partly regulated by thyroxin. The whole process can be rapidly speeded up by treating a tadpole with an exogenous source of thyroxin. Immediately, morphogenesis begins to occur such that by day 9 legs and forearms are present on the treated tadpole (insert, Figure 15), whereas its control has none. The question thus arises, what would happen if the thyroxin or a thyroxin-like hormone had been mutated such that none or too much active hormone was secreted? What if it happened at the wrong time? If the results of various mutations studied in *Drosophila* during metamorphosis is any indication the results would be deleterious (most likely lethal).

In normal morphogenesis the collagenase must be uniformly secreted along the length of the tail, so as to allow for the resorption (removal by digestion) of collagen in a uniform manner. Diffusion from a central core of cells for several millimetres could not account for this. As it turns out there are very elongated thin processes of cells that extend between the alternating collagen bundles. It is from these that the collagenase is secreted, such that the skin of the entire tail region resorbs relatively uniformly.

Resorption of the tail and development of the legs and forearms must be meticulously co-ordinated. If the tail disappeared too quickly before leg formation then the tadpole would be immobilized, and thus most likely consumed. At the same time, the tadpole changes from an obligatory aquatic respiratory system to that of an air-breathing amphibious existence. Thus if the air-breathing capacity developed before the leg developed, but the tail had already been removed, the animal would drown.

Therefore, many enzymes, collagenase being only one of them, must be activated or deactivated in a precise concerted fashion during this morphogenesis. Alterations caused by mutations are deleterious. This is especially true with collagenase not only when referring to morphogenesis but also in body metabolism as a whole. For example, "Liotta *et al* (1980) have been able to show a relationship between the amount of collagenolytic activity released per cell and the metastatic potential of two different malignant mouse lines, the B-16 and the T-241 sarcoma, and also a culture of human breast carcinoma cells. . . .At least some neoplastic cells with metastatic capability manufacture the type IV — specific collagenase that facilitates their passage through the capillary wall and may have the capacity to degrade stromal collagen or induce the host fibroblasts, via secreted products, to secrete a type I — degrading collagenase."[55]

The effects of mutations upon the morphogenesis of humans is also becoming well documented. L.B. Holmes has stated in the **New England Journal of Medicine** that, "two percent of new born infants have a serious malformation. Malformations are the most common cause for hospitalization of children in North America. The two categories customarily used for classification are multiple malformations and single isolated or localized malformation. Of the categories the single, localized, major malformations are the most common. . . .It is appropriate to regard these localized hereditary malformations as inborn errors of morphogenesis."[56]

Baraitser and Winter concur with him stating, "Genetically detrimental conditions are individually rare but collectively they contribute significantly to the 2–3% of children born with malformations. If those conditions with a later age of onset are added to the total, it is not surprising that there are between 2000 and 3000 conditions listed by McKusick in his catalogue of Mendelian Inheritance in Man."[57] Mutations that affect human development are clearly harmful.

Another recent well-publicised result of mutations affecting morphogenesis are those relating to alterations in the Biothorax Complex (BX-C) of *Drosophila*.[58] By mutating three genes in this region a duplication of the mesothorax occurs (with no development of a metathorax), and results in the presence of four rather than two wings. However, the mutant cannot fly. There are other examples as well in which an organ develops in a wrong position:[19] Antennapedia results in legs growing where the antennae usually develop, Ophtalmoptia results in wings developing where eyes should, and Proboscipedia in which legs develop at 25°C and antennae at 17°C where normally a proboscis would form. Ayala states that the expression of these mutations is usually weak, and affects only certain sensitive cells and thus while gross deformities occur, they are not lethal. However, in some cases where the mutation completely abolishes a gene's function and affects most or all cells, the mutation is usually lethal to the embryo.

Three points should be made concerning these observations:

i. The alterations are not useful, that is, the four-winged *Drosophila* can't fly;

ii. The alterations are not new or 'de novo' structures, but are simply duplications or displacements (at the expense of some other organ or segment); and

iii. The *Drosophila* remains just a *Drosophila*, albeit a debilitated one.

In conclusion many organisms go through a complex morphogenesis including some insects, amphibians, and marine animals. Mutations are known to dramatically affect development, but the changes are detrimental to the organism.

(5) The leaf-curling spider is a good example of design at the level of habit or intelligence (Figure 16).[60–61] It is native to southern Australia and builds webs that are three dimensional — a front circular web, with runners protruding back from the face of the web to hold it taut. This spider, weighing only four grains (approximately 0.25 grammes), then searches for a flat, usually dry leaf. It draws this leaf, or even a snail's shell [up to 24 grains (approximately 1.55 grammes) in weight] two to three feet up into the centre of its web. The leaf is curled into a cone shape and secured into the web. The spider lives inside the cone.

If grass is thrown onto the web, the spider quickly removes it, and returns to its leaf home by a rapid retrieval line. It also waits for prey with its feelers gently touching the web, and can differentiate between a false vibration of the web by the wind or human touch from a fly caught in it.

Even in this simple animal there is a complex life pattern. How does it 'know' how to spin the web, with a correct tautness, and thickness of silk, and that it must be a three dimensional web so as to hold the leaf? How does it know how to lift a leaf up into the web or to remove grass from its

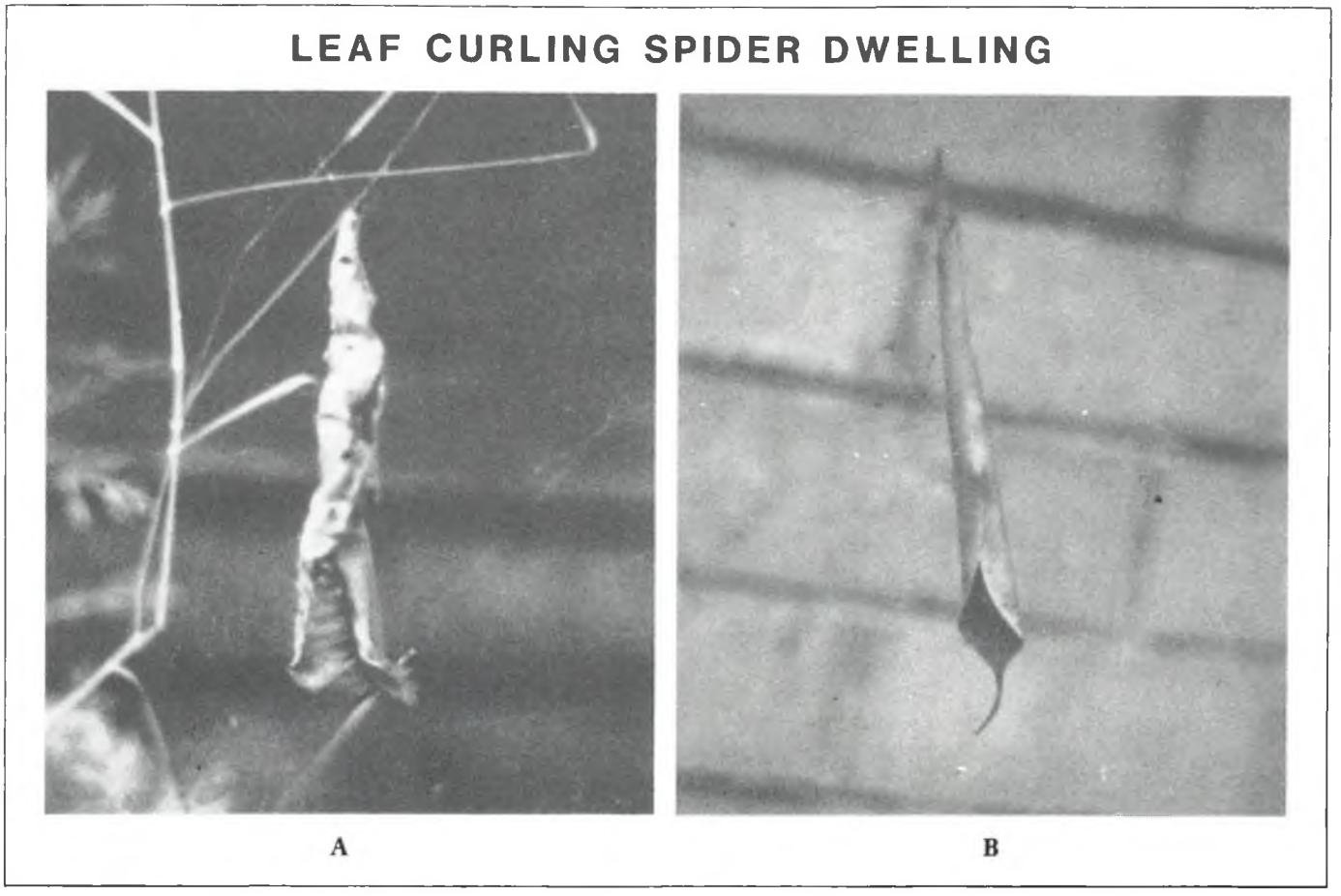

Figure 16. *Design at the level of habit or intelligence — the leaf curling spider. (a) and (b) are two different dwellings produced by these spiders. Each was suspended in a web approximately one metre above the ground. In (b) the spider can be seen partially obscured within the leaf dwelling place.*

web? All organisms express this sort of complexity.

Mutations can cause alterations in habit or intelligence. But they are deleterious aberrations. For example, the mutant 'stagger' mouse is a good example of this. In this mouse there is a defective conversion of fetal cell attachment proteins (CAM) to adult ones.[62-63] These proteins are necessary for proper cell adhesion and development. The 'stagger' mouse's brain does not develop properly due to the mutation and the result is ataxia. In humans, children with the disease phenylketonuria or Lesch-Nyhan Syndrome are sometimes prone to self-mutilation. Thus it is clear that mutations can degenerate habit or intelligence, but it is not so clear that they can generate it.

(b) Optimized Structures

Optimize (Webster's Dictionary) means "to make as perfect, effective, or functional as possible." All living organisms appear to have optimized structures.

(1) The Genetic Code

i."The groups of codons which correspond to the same amino-acid in the genetic code (synonyms) are compared to theoretical codes constructed so as to resist best to the effects of mutations. The analysis shows that the genetic code presents synonymy structures which are optimized against translation error. . .The assignment of an amino-acid to a given codon is not at random, similar amino-acids correspond to neighboring codons."[64]

ii."It can be shown that the different amino acids translated in the proteins, except the particular case of SER, obey a logical code for optimization of resistance to mutation effects."[65]

(2) The building blocks-nucleotides and amino acids. "Numerous effects of nucleotide pool imbalances have been described in phage, bacterial or yeast

test systems. Thymidylate starvation leads to induction of an error-prone repair system in *E. coli* and induction of mitotic recombinations in yeast. Excess thymidine is mutagenic to both bacteriophage T-4 and yeast."[66] Thus, there are optimum nucleotide pool concentrations in cells, and these can be altered by mutation. But the effect is harmful.

Amino acids are all of the L form in living organisms (there are a few exceptions, like some proteins in the bacterial cell wall). Shimizu comments, "The optical activity of the proteineous amino acids used by the organisms on the earth are restricted to be of L type and that of the nucleotides to be of the D type. This is one of the distinguished features of the terrestrial living system as well as the uniqueness of the genetic code etc.. . ."[67] A mixture of L and D, or even one D form would be deleterious in that proteins containing any D forms could not form similar helixes as the ones containing all L forms. In carbohydrates and DNA it is exactly the opposite, where the monosaccharides are all D forms. It is thus optimal for living organisms to use only one form. Yet, most if not all prebiotic mechanisms for making animo acids yield racemic mixtures (for example, Miller's experiment).

(3) Proteins

i. "It is generally accepted that the disturbance of the protease-protease inhibitor balance can lead to protease mediated tissue destruction."[68] A good example of this is the disease in which the $\propto 1$ proteinase inhibitor (a proteolytic enzyme inhibitor) concentration is deficient (due to a mutation) in the lung.[69] People with this disease often contract emphysema, due to excessive proteolytic degradation of lung tissue.

ii. "The source of antibody diversity is now evident. Just as shuffling an alphabet with a finite number of letters can yield an infinitely rich language, so can rearranging V, J, and D genes yield virtually unlimited numbers of antibody specificities. And as if the process of rearrangement were not enough, somatic mutation of V genes and imprecisions in the joints of fused V-region genes add to the diversity."[70] Clearly, antibody formation is a precise, regulated, optimal procedure where both the heavy and light chains are produced in equimolar amounts. The diversity of specificity is due to rearrangement of various genes, and this can be affected by somatic mutation. The effects of the mutations vary from altering the original specificity of the antibody to causing a non-functional antibody to be expressed.

"During human development there is a switch from fetal to adult haemoglobin, reflecting the differential expression of fetal ($G\gamma$ and $A\gamma$) and adult (β and δ) globin genes. Mutations that inhibit this switch produce variants of the syndrome of hereditary persistence of fetal haemoglobin (HPFH)."[71] Haemoglobin production is coordinated and optimized in several ways. Firstly, the oxygen carrying molecule is made up of four peptide chains derived from two different genes (eg. $\propto$ and β). Expression of both genes must be equimolar or an imbalance due to mutations results in a disease state (Thallasemia). Secondly, different genes are expressed at different stages of development of the human being, that is, there are embryonic, fetal and adult haemoglobin molecules. Alterations in the time of expression of these chains, due to mutations, results in various disease conditions (as stated above).

(4) Structures

Virtually any structure or organ that is analysed in any living organism shows optimized function.

i. Bones are optimized by three criteria:[72]

a. They are tubes — thus resist bending;

b. Hollow — less force needed to move them; and have

c. High tensile strength — they resist cracking under tremendous forces of stress.

Mutations mentioned earlier affecting the collagen or proteoglycan of bones can render the bones virtually non-functional.

ii. Tendons are composed of separate fibres to resist tearing, whereas a uniform material would tear more easily.[73] Mutations in collagen structure can affect tendon function adversely.

iii. The intervertebral disc annulus has laminae of collagen fibres at 65° angle tilt alternating in opposite directions. This gives increased strength.[74] The nucleus (within the centre of the intervertebral disc) contains unordered collagen fibres and hydrated glycosaminoglycans. Therefore, in humans when vertical pressure is applied to the nucleus, it spreads the pressure horizontally and equally in all directions to the annulus. This is an efficient mechanism for dispersing impact pressure on the spine. However, it requires correctly assembled intervertebral discs which in turn are dependent on functional, strong collagen fibres and polyanionic glycosaminoglycans. Mutations (mentioned earlier) to either of these macromolecules are deleterious.

Design and optimization are evident throughout all living organisms. In conclusion:

— No biomolecule functions independently of other molecules. There are complex optimized interrelated systems of interaction between the estimated 50,000 to 100,000 different types of proteins, as well as nucleic acids, fatty acids, polysaccharides and amino acids, etc.

— There are many examples of co-ordinated multiple organ functions (eg. bombardier beetle) that cannot be explained by individual rare mutational changes. The various organs require the complete functional capability of the other organs if they are to work at all. This is even seen at the molecular (ATPase), and the individual organ level (aorta).

— Examples are plentiful of the degenerative effects of mutations on cellular design, optimization and function, but there are few if any that improve them.

3. Probability

Sir Julian Huxley estimated that the evolution of the horse took over a million mutations.[75] Winchester has stated that 99% of all mutations are harmful,[76] while Dobzhansky has stated that most are actually lethal.[77] It is also apparent that mutations are a rare event. Thus, what is the likelihood of rare mutations, that are rarely (if at all) good, occurring at least a thousand times in one type of organism?

In answer to this question Cribbs and Barrows have stated, "We based our model upon a bacterial population in which one cell in every billion cells mutated. The probability of a mutation being harmful in some way was 99.99%." (This is acceptable to many evolutionists.) "The remaining 0.01% was divided into neutral, reversion, and beneficial mutations. Here we were assuming that beneficial mutations could occur in our bacterial populations. As our model demonstrates, within valid parameters, mutations result in an inevitable lethal genetic burden on the progeny of the bacterial cell in which the mutations occur. Offspring always reach the absorbing state of death, and the population as a whole suffers from 'dirtying of the gene pool'."[78]

G.G. Simpson, an evolutionist, has stated that, "the probability of five mutations in the same nucleus would be $1/10^{22}$."[79] From this he reasoned that "With an average effective breeding population of 100 million individuals and an average length of generation of one day, again extremely favourable postulates, such an event would be expected only once in 274 billion years, or about a hundred times the probable age of the earth. . .unless there is an unknown factor tremendously increasing the chance of simultaneous mutations, such a process has played no part whatever in evolution." And this is not even taking into account harmful versus beneficial mutations.

The probability of successive good mutations could be analogous to the likelihood of an escape of a wild animal from an open air cage. Most of these types of cages have a double set of doors, so that if per chance an animal gets through the first door (a rare event) it is very unlikely he will get through the second as well before the caretaker notices and catches him. The first door is like a supposed rare "good mutation", the second door (and really we should expand the area in front of it to a cage the size of the first cage) is a second supposed rare "good mutation". The caretaker is like natural selection who catches (or removes) the animals before they escape through either door. But one might say, "It is still possible that an animal might escape." Yes, it is possible, but not probable. Expand the number of doors to one thousand and it becomes very improbable.

Hoyle and Wichramasinghe (both evolutionists) have stated that the probability of evolution from simple particles to present-day complex life is $1:1 \times 10^{40000}$.[80] Thus, they argue that evolution, at least from simple molecules to simple-celled organisms, must have occurred somewhere in space and then arrived here as spores. Crick, the Nobel Laureate, subscribes to this concept too.[81] Of course, this only removes the problem (prebiotic mechanisms of origins) to a place where it cannot be observed. But still they acknowledge that it is very unlikely that random aggregations of nucleotides and haphazard rearrangements (mutations) of these could account for the development of the so-called simple single living cell.

It is interesting to note that it has been estimated that the likelihood of two identical fingerprints occurring is $1:3 \times 10^{42}$. From this the authors conclude, "Under the circumstances it is impossible to offer decisive proof that no two fingers bear identical patterns, but the facts in hand demonstrate the soundness of the working principle that points from two different fingers never are identical."[82] Courts have used fingerprint evidence as proof enough to convict people. Why not apply this criterion to the probability of mutations being the source of macroevolution (that is, reptile to bird)?

4. Natural and Other Types of Selection

There are several ways in which organisms may be 'selected for' and allowed to continue to contribute to the gene pool of their group, and for other organisms to be 'selected against' so that they

are removed (eliminated) and no longer contribute to the gene pool:

(a) Selection of healthy (fit) versus unhealthy (unfit or less fit) organisms (that is, 'Natural Selection');
(b) Selection of normal traits (not mutated) under different environmental conditions;
(c) Non-discretion selection;
(d) Selection for cripples; and
(e) Artifical selection.

There are three possible views concerning the outcomes or results from a selection process:

— It eliminates the unhealthy, defective organism; or

— What survives, survives; or

— It results in better adapted, and even new innovated structures.

It has been postulated by evolutionists that mutations are the source of all new genetic material and that 'Natural Selection' operates on these to produce evolutionary advancement. Gould has stated, "No one denies that natural selection will play a negative role in eliminating the unfit. Darwinian theories require that it create (that is, mutate) the fit as well. Selection must do this by building adaptations in a series of steps, preserving at each the advantageous part in a random spectrum of genetic variability."[83] (insertion mine)

On the other hand, creationists would argue that the overall effect of any type of selection generally results in the elimination of harmful genetic alterations (mutations), that no 'new' structures or increased function (that is, adaptive potential) occurs by selection, and that organisms always remain the same type of organism before and after selection (Figure 17).

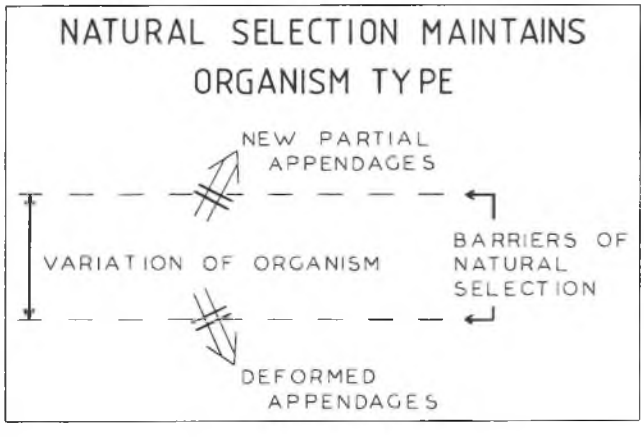

Figure 17. *Natural selection maintains organism type. Deformed (mutated) structures are eliminated as well as so-called partial 'de novo' structures.*

What do the observations on the various types of selection reveal?

(a) Selection of the Healthy (Fit) Versus the Unhealthy (Unfit) Organisms (that is, 'Natural Selection')

As Gould stated, "No one denies that natural selection will play a negative role in eliminating the unfit." It is known by actual observation that many (virtually all) mutations decrease the 'fitness', that is, reproductive capacity, weight, resistance to disease, etc., of animals and plants.

The examples are plentiful, and the severity of each can be divided into lethal (death before puberty [DBP] 100%), semi-lethal (DBP $>$ 50% but $<$ 100%), sub-vital (DBP $<$ 50% but $>$ 0%), and normal (DBP$\sim$ 0%).[84] Many of the homozygous recessive lethal conditions when in the heterozygous condition cause the animal to be affected, but it is not lethal. Examples of the homozygous recessive lethal condition are:[85]

(1) Bulldog Calves in Dexter cattle (result: achondroplasia — extremely short legs and compact bodies);
(2) Creeper Fowls in American Creeper and Japanese Bantam chickens (result: shortened limbs);
(3) Pelger Rabbit (result: stunted growth).

These animals do not breed properly, die young, and therefore do not contribute to the gene pool. They are 'unfit' and are eliminated under conditions where normal animals would flourish.

If these sorts of mutations occurred to animals in the wild their fate would be even swifter and more sure. Cripples simply don't survive, except under unusual (artificial) conditions — see below.

Thus the effect of this sort of selection would be to stabilize a deteriorating gene pool, in that many mutations would be removed (although not completely if they are recessive). Also major structural alterations would be removed that might have been considered the beginnings of 'de novo' appendages. Thus, this sort of selection would hamper evolutionary advancement but would be complimentary to the concept of creation. It is by far the greatest type of selection in nature, the others listed are minimal in comparison.

An interesting variation of natural selection of the 'fit' versus the 'unfit' occurs during the competition between the beetles *Tribolium confusum* and *T. castaneum*.[86] Under normal conditions *T. confusum* is eliminated from the population. However, an eye colour mutant of this species appeared (in one experiment) to be more 'fit' in that it survived and eliminated the members of *T.*

castaneum. However, the mutant had a reduced overall fitness or adaptability compared to the normal *T. confusum*. As it eliminated the *T. castaneum*, the survival ability of *T. confusum* also decreased, and resulted in its elimination. Thus, a mutation that appeared to make an organism more 'fit' still debilitated that organism, and eventually resulted in its extinction.

Another example of this is the 'sex-ratio' chromosome in *Drosophila*.[86] A male fly with this defect produces only female offspring. Thus within a given interbreeding population this could lead to increasing numbers of females and decreasing numbers of males, and could eventually lead to extinction of the population. Thus, while some might argue that the 'sex-ratio' mutant is more 'fit' because more females than males are produced, the overall effect of this mutation is harmful to the population.

(b) Selection of Normal Traits (Not Mutated) Under Different Environmental Conditons

Probably the best examples are the *Betularia* moth and Darwin's finches. Both have been called principal examples of evolution and yet neither is such an example (at least when discussing the concept of transmutation from one type to another, that is, fish to amphibian).

The *Betularia* moth is present in a white or black variety.[87] During the Industrial Revolution of the 1880s the trees around London became covered with black soot. Up until that time they had been varicoloured light bark. Therefore, before the onset of soot deposit, white moths, which blended more readily with the bark of the trees and were therefore less visible to birds, were probably in dominance over the black moths. With the onset of the Industrial Revolution the reverse occurred. Since the improvement of air quality in London in recent times, the white variety is again increasing in numbers.

Is this evolutionary change? The answer is no. Black and white moths were there in the beginning, and black and white moths were there in the end. It is only the ratio in the population which has changed.

Also, since the various varieties of this moth freely interbreed, the gene pool maintains heterogeneity for the body colour. However, if the black and white varieties did not interbreed, it is very possible that this species of moth at least in the London area would have become virtually extinct, first by removal of the white moths and then the black ones. The ability to interbreed increases the ability to adapt in a group of organisms. And this simply increases the likelihood of survival of that 'kind'.

Darwin's finches are also just another example of selection within a kind of organism. Although in this situation it is not based on just one trait, but a series of traits, and the result is not really distinct groups (species) of finches but rather intergrated groups that still remain just finches (see speciation).[88]

Thus certain environmental conditions can encourage the enhancement of certain attributes, and organisms with the greater heterozygosity of genetic material are the ones most likely to be able to adapt. But organisms still remain the same type before selection and after it. Thus, while this is compatible with variation within kinds in the creation philosophy, it argues against the transmutation of the evolution philosophy.

(c) Non-Discretion Selection

This is where the organism selected for elimination is indistinct from other ones, and is simply removed by 'lot'. An example of this would be where a hawk catches one of several identical healthy mice running in an open field. This sort of selection would not help evolution theory because there is no orientation for selecting for increased complexity, but it presents no conflicts with the creation model.

(d) Selection for Cripples

This is probably the most controversial type of selection. Can a mutated (that is, crippled) organism be selected for survival rather than the non-mutated one? The answer is yes. But can this be used as an example of evolutionary advancement, that is, as Gould said to "Create the Fit"? The answer is no, for several reasons:

— The organism **never** changes type, that is, mutated and selected *Drosophila* remain *Drosophila* (even after thousands of generations under enhanced mutation rates); and

— Mutated organisms may be selected for under unusual (usually artificial) circumstances but their overall functional capacity (adaptive potential) appears to be always decreased.

A hypothetical example is shown in Figure 18. An organism incurs a mutation in one of its vital metabolic pathways (M), which results in no uptake of precursor material. However, it is still able to make the essential biomolecule by using a secondary pathway (S). Both it and normal organisms are placed in media which contains a toxin taken up in the main pathway. The normal organism dies but the mutated one lives. Is this evolutionary advancement?

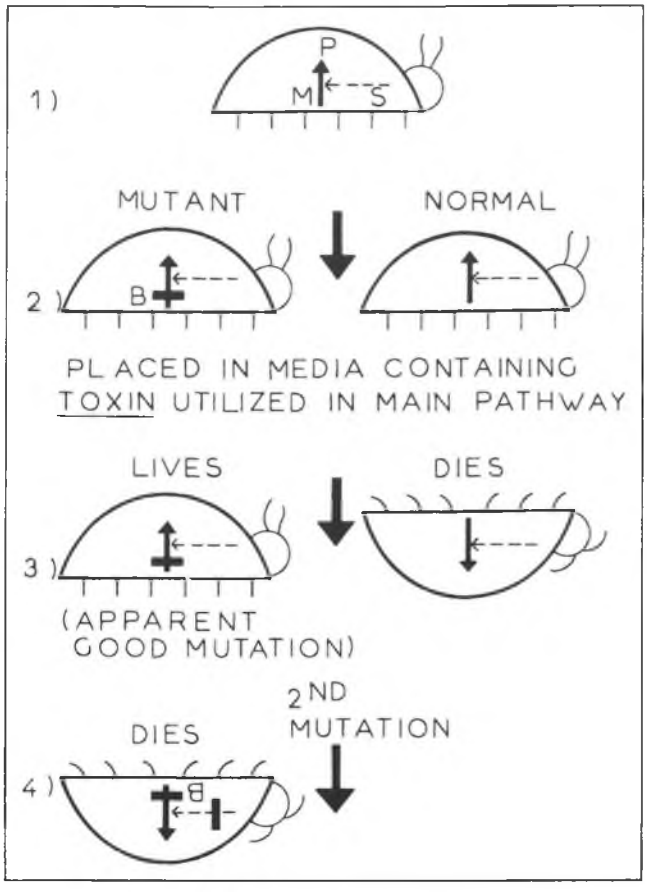

Figure 18. *Natural selection can select for survival of a mutant (defective cell or organism) rather than a normal cell or organism under certain conditions. This results in an "apparent good" mutation. Further mutations can compound the effects of this mutation and result in the death of the cell or organism. Main Pathway (M), Secondary Pathway (S), (B) block caused by mutation, (P) essential metabolic product.*

The answer here is no for several reasons:

— Gene Mutational Load — if the organism mutates again in the secondary pathway it will die no matter what environment it is in (that is, the compounding effect of multiple mutations);

— The main pathway is defunct, thus the useful information content within the organism is decreased (that is, functionality is decreased), the overall vitality of the organism is decreased and when it is placed in a non-selective environment it is rapidly overtaken by non-mutated ones. Thus it possesses less adaptive potential than the non-mutated organism.

Examples of this are numerous but three well known ones will be explored:

(1) Drug or Chemically Resistant Organisms

i. Animal — methotrexate.[89–90] This drug inhibits dihydrofolate reductase, a key enzyme in folate metabolism which is necessary for nucleic acid metabolism. But some cells become resistant to the drug. There are three ways that this occurs. The first is by a mutation in the enzyme that alters its structure such that the high affinity for methotrexate is lost, but enzyme activity is also decreased. A second mechanism is by an alteration in membrane permeability (due to a mutation). And thirdly by amplification of the gene for the enzyme. This can result in up to a 400-fold increase in enzyme and 5% of the total protein synthesis of the cell. However, with increased gene amplification, cell growth decreases, such that when the altered cells are removed from methotrexate and placed among normal cells they are rapidly overgrown. Another problem is that some cells lose the amplified genes after removal of methotrexate, while others remain stable.

Therefore, while it is remarkable that these cells can sometimes adjust (by gene amplification) to a toxic environment, it adversely affects them. Mutants which survive under these conditions are not advanced, but simply survive, and are actually debilitated.

Other examples of gene amplification occurring when an organism is placed in a toxic environment are the bacterium *Salmonella typhimurium* in which genes coding for histidine are amplified, and also other bacteria where glycyl transfer RNA genes are amplified.

ii. Plant — 5-methyltryptophan.[91] This drug, an antagonist to tryptophan, inhibits anthranilate synthetase, which is normally feedback inhibited by tryptophan. Since this enzyme is involved in the production of the indole ring, inhibition of it results in decreased production of tryptophan, such that most of the cells die. However some cells live because the enzyme has been mutated such that it is no longer inhibited by the drug, and thus tryptophan continues to be produced in excess.

Thus the normal cells die, but the mutants live. However, this cannot describe evolutionary advancement for the cells have become unregulated because they are no longer feedback inhibited.

A similar example to this is where fast-growing tumour cells are placed in culture with normal cells. The tumour cells will outgrow the normal cells and thus the normal cells will die. But is the tumour cell a better or more advanced cell? No, it is a dedifferentiated unregulated cell, the result of a process of deterioration.

iii. Bacteria — Isoniazid.[92] *Mycobacterium tuberculosis* bacteria that have become resistant

to this drug experience a loss or reduction of catalase activity, as well as a deficiency in dehydrogenase and urease.

"It was observed that nearly all of the isoniazid-resistant mutants developed *in vitro* or isolated from patients under treatment with isoniazid have markedly reduced virulence for the guinea pig. There is a close correlation with the degree of isoniazid resistance of the tubercule bacilli and the reduction of their virulence for the guinea pig."[92] Another way to say that is that there has been a decrease in the bacterial cell activity and function.

Bacteria have also shown resistance to the drug streptomycin.[92] This results in an increased production of enzyme for the utilization of salicylates.

Thus, drug resistance can result in an imbalance of enzyme activities.

(2) Monoclonal or Fused Cells

Fusion of two different,[93] or even three different types of cells[94] can result in cells that express characteristics of all two or three. This technique has been invaluable in developing monoclonal antibodies. In a typical experiment mouse myeloma cells (which have been previously selected so as to not secrete antibody and lack the HGPRT enzyme) are fused with immunized mouse spleen cells using sendai virus or polyethylene glycol as the fusion agent (Figure 19). The resultant hybrids express characteristics of both cell types; they rapidly divide like the tumour cells, and express antibody like the spleen (B) cells. After fusion the non-fused cells must somehow be removed from the hybrids (spleen cell/myeloma cell). This is easily done because non-fused spleen cells simply don't live well under present *in vitro* conditions. The unfused myeloma cells are eliminated by placing the cells in a medium which contains aminopterin. This drug blocks the major pathway of nucleotide synthesis. Most normal cells would still live in the presence of the drug by utilizing a secondary pathway to metabolise the nucleotides, but because the myeloma cells lack HGPRT activity the secondary pathway is also blocked so these cells have no way of making nucleotides and thus they die. This is somewhat analogous to our

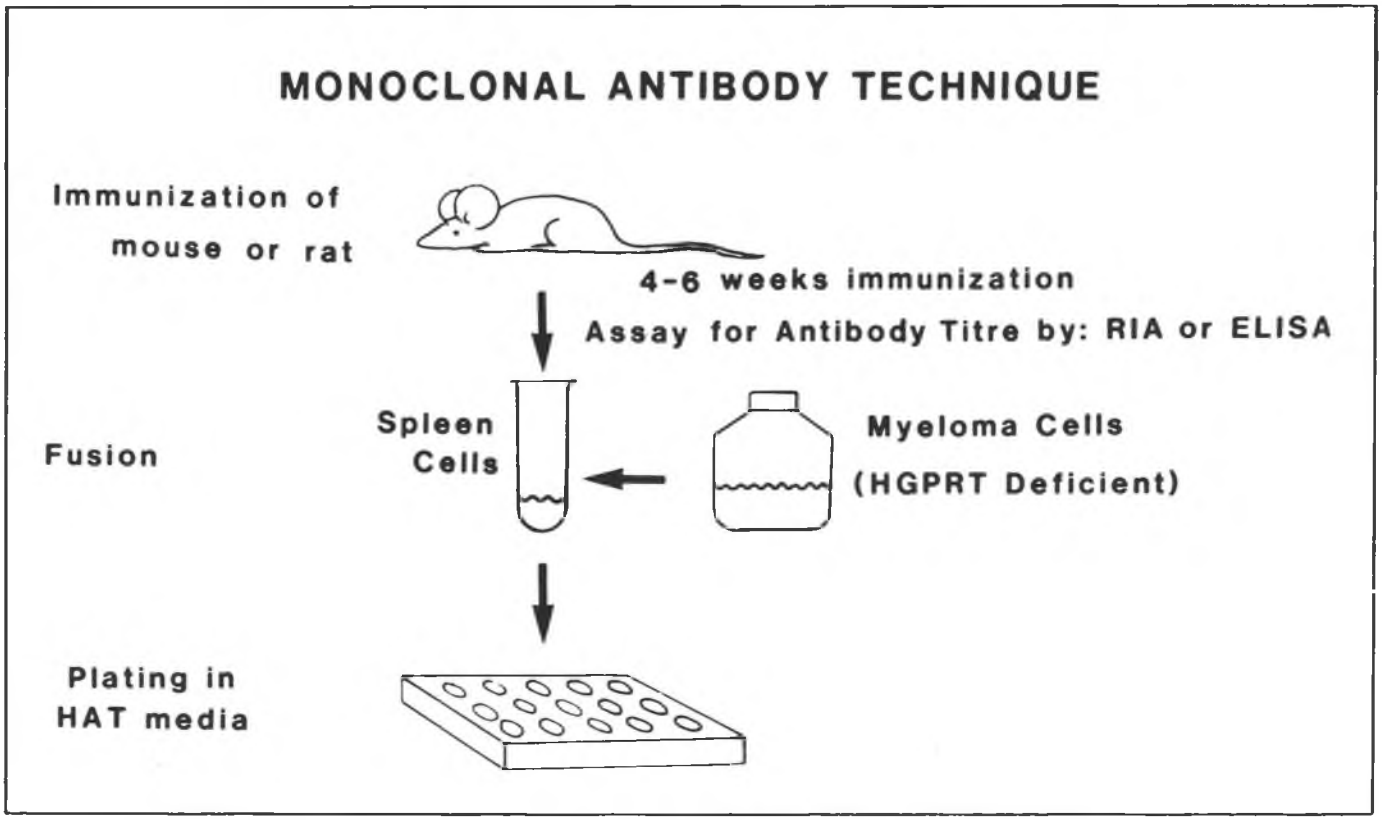

Figure 19. *The production of monoclonal antibody-producing hybrid cells — selection of a 'crippled cell'. Hybrid cells are produced by fusing mouse or rat spleen cells with myeloma cells using polyethylene glycol. Non-fused myeloma cells are eliminated by the addition of HAT medium (HAT = hypoxanthine, aminopterin, thymidine). Unfused spleen cells passively die off and only hybrids are able to survive. RIA = Radioimmune Assay, ELISA = Enzyme Linked Immunosorbent Assay. Both are used for detecting specific antibody production. HGPRT = hypoxanthine-guanine phosphoribosyl transferase.*

hypothetical example (see Figure 18).

Thus does this fusion/selection process result in an evolutionary advancement — the hybrid cell? Again, it can be said that it really says nothing directly about transmutation from one species to another. The hybrid cell is also an unregulated, unstable cell type. As with many cells that contain excess DNA, these hybrids begin eliminating, somewhat randomly, the excess DNA. In one study using a human × human cell fusion,[95] after six weeks, the number of chromosomes per cell varied from 31–100 (the expected number would be 46/cell × 2 = 92). The highest percentages were between 41–45 (20%), or somewhat similar to what a single normal cell would carry. Thus, these cells are highly unstable genetically. This probably affects their viability, in that shifts in pH appear to kill them more easily than normal cells. Also, if they are placed in normal media with non-fused myeloma cells, the hybrids appear to be rapidly eliminated by an overgrowth of the myeloma cells. They are dedifferentiated in that they still retain the characteristics of an unregulated cancer cell. Thus, this does not support evolutionary advancement, but rather that there is a fixity of cell types and that debilitation of these cells occurs by mutations or other alterations such as fusion techniques. This is true even though these cells can be selected for survival rather than the non-mutated or non-fused ones.

(3) Crown Gall Disease

This disease is caused by the bacterium *Agrobacterium tumefaciens*.[96] The bacteria infect the root cells of plants or trees, especially almond trees. It transfers plasmids which contain genes coding for various factors, to the root cells and this causes the root cells to transform. The "symptoms include roundish, rough-surfaced galls, several inches or more in diameter, usually at or near the soil line. . .as the disease progresses, plants lose vigour and may eventually die."[97] This "uncontrolled cell division of these tumorous plant cells continues even after the bacteria are eliminated."[98]

Two pertinent conclusions can be drawn from the observations of this disease. The plant cells which have received foreign genetic information (plasmid) become unregulated and dedifferentiated (tumour cells). (Viral insertions into cells cause similar problems, for example, oncogenes.) Tumour cells become the predominant cell type in the nodules and this results in the death of the plant. Therefore, even though crippled cells survive (tumour cells) in the root temporarily, this causes the overall destruction of them and the rest of the plant. Thus, the 'fitness' incurred on the crippled cell is only an apparent one.

A final comment about the process of selecting for mutant cripples is that it actually compounds or accelerates the effects of mutations within the population, for example, breeding of dogs for certain mutant characteristics such as the bulldog's flattened face.

(e) Artificial Selection

Edward Blyth stated in 1835, 'the same law (natural selection), therefore, which was intended by Providence (God) to keep up the typical qualities of a species, can be easily converted by man into a means of raising different varieties (artificial selection). . .they (referring to adaptations for protection and adjustment to the environment) are among those striking instances of design, which so clearly and forcibly attest the existence of an omniscient great First Cause."[99] (insertions mine) It is still true that artificial selection can be used to produce widely differing varieties of the same type or kind of organism, like dogs, cats, and pigeons. This ability to select such diverse characteristics reveals how rich in diversity is the gene pool of various types of organisms. Yet, using artificial selection, never has one organism ever been transmutated into another one. This supports strongly the creationist perspective that organisms were created as distinct groups and reproduce 'after their kind'.

It is intriguing to note that Charles Darwin took the observations of Blyth of artificial selection and extrapolated them to mean that transmutation could occur. He also commented that he had no direct empirical proof of this sort of change (that is, variation) resulting in transmutation (for example, reptile to bird).[100] Thus, he held on to his theory even when he lacked empirical observations. R.A. Fisher, another well known evolutionist, in correspondence with J.S. Huxley (1930) about a book he (R.A.F.) had just written said, "As it is there is surprisingly little in the whole book that would not stand if the world had been created in 4004 BC, my primary job is to try to give an account of what Natural Selection **must** be doing, even if it had never done anything of much account until now."[101] (R.A.F. emphasis on **must**.) The implication is that his observations could

have been compatible with a young earth creationist viewpoint.

It is also important to realise that even artificial selection can be misused to promote and retain harmful characteristics in a population. Examples among the dogs are: the collie has an overshot mouth, boxers have undershot mouths which results in crowded teeth and this can result in dental diseases. Bulldogs have an overlong soft palate which obstructs their airway. The Pekingese' eyes are unprotected, lacking a proper bony socket, and are thus more subject to eye injury. Dachshunds have excessively long backs and are subject to spinal degeneration.[102]

Wysong makes an interesting comment, "There are numerous examples of freakish mutations in the biological world. There are two-headed fish, one-eyed fish, siamese twins, bull dog calves and thousands of artificially produced fruit fly monstrosities. Mutations of this type, and those listed by Montague above, seem obviously not beneficial. But hasn't man produced improved varieties? Yes, man has produced through mutations and carefully controlled selection, 'improved' organisms that benefit man. Seedless grapes, for example, are easier for people to eat, but hardly would seedlessness help the grape in the wild. A short crooked-legged sheep, the Ancon breed, produced through genetic manipulation, is unable to jump fences and maintains weight better than the normal more ambulatory breeds. However, the fertility of this ram is markedly reduced, and hardly would the mutated weakened legs aid the ram in escaping from predators in the wild state. All mutants, as Nilsson and others implied above, are weakened for existence in the wild state; mutant 'improvements' seem to be only in reference to their value to man."[103] Beadle, an evolutionist, responds in a similar fashion when he calls domestic corn, a 'biologic monstrosity', that would not survive in the wild.[104] Lammerts, from his work with roses, noticed that "Mutations can alter only the various phases of the basic varietal pattern expression; the pattern itself is not changed. Truly unique and outstanding varieties such as Peach, Charlotte, Armstrong, or Queen Elizabeth would never result from the accumulation of mutations. . .An interesting feature of this work is. . .all, without exception were weaker than the variety originally irradiated. . .or have a reduced fertility, in terms of either the percentage of good pollen or number of seeds produced per plant."[105]

Thus, artificial selection reveals the tremendous diversity available in various types of organisms. But it also reveals the inherent fixity of each type or kind. And as with the selection of cripples, it can be used to promote mutational characteristics, but these are never really helpful to the animal.

In conclusion, by and large, natural and other selections remove debilitating, harmful mutations from populations of animals. In a few unique (and usually artificial) circumstances a mutational characteristic can be selected for in the survival of the organism. But it can be shown that invariably these mutants are debilitated or less functional (that is, having decreased adaptive potential) than the non-mutated ones. Natural selection processes do not support evolutionary transmutation. But even if this conclusion is not accepted by some, it must still be clearly understood that no empirical observations have ever been made that actually reveal the action of natural selection upon a series of mutations to transmutate one type of organism into another type (for example, reptile to bird).

5. Gene Mutational Load/Gene Pool Size (Heterozygosity/Homozygosity)

Gene mutational load has been defined as "the proportion by which the fitness of the optimum genotype is decreased by deleterious genes as expressed in lethal equivalents or 'genetic deaths'. In the human population, the genetic load was estimated by Muller to be four lethal equivalents or recessive genes that are lethal as homozygotes."[106] Most scientists would agree that all organisms have been and are affected by mutations and thus by gene mutational load. But does it play a significant role concerning evolution or creation? There are several concepts to consider.

Firstly, a large portion of lethal or deleterious genes are homozygous recessive traits. Concerning the ability of a population to rid itself of recessive mutations, Ayala and Kiger have stated, "The frequency of the recessive allele for albinism in Norway is about 0.01. Assume that a eugenic goal is established to eliminate the allele from the population by sterilizing all albino individuals. It would take 100 generations to reduce the allelic frequency to half its present value and 9900 generations to reduce it to 0.0001. Eugenic measures are inefficient in the case of recessive alleles."[107] Thus, it is virtually impossible to remove them from a population completely. The frequency may wax and wane due to various selective pressures, but they are unlikely to be removed from the population. Therefore mutations become 'fixed' in populations.

Secondly, mutations are known to spontaneously arise, and many of these would be homozygous recessive traits. These sorts of mutations are known to affect any one of the human 22 somatic and 2 sex chromosomes, and probably any locus on each (although this is not yet proven,[108] but is indicated from the large number of examples of altered human karyotypes).

Therefore, it is conceivable and even probable that the number of mutations present within the genetic makeup of any organism is increasing. And this increase is passed from generation to generation. This is in accord with the Second Law of Thermodynamics which states that the overall information content of anything decreases with time. Knowing that mutations are virtually, if not always, harmful, and that the number of them is increasing with time in living organisms indicates that they (organisms) are in a state of deterioration. Natural selection may act as a retardant to this process but the overall effect is a downward slope (Figure 20).

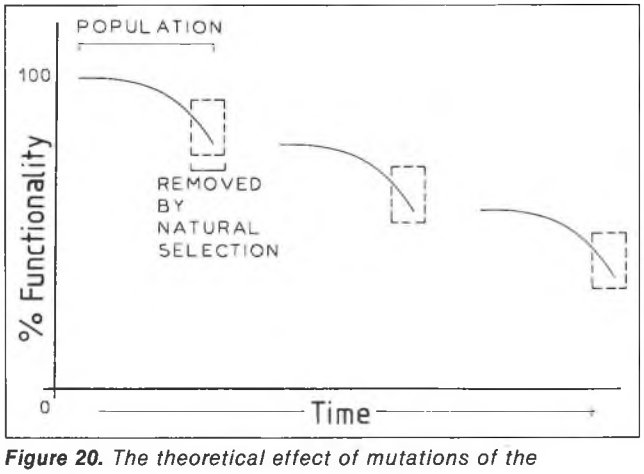

Figure 20. *The theoretical effect of mutations of the functionality (or adaptive potential) of a population of organisms.*

Man's lifespan as recorded both in the Bible[109] and other secular sources (Babylonian)[110] support this concept. The first men recorded in the Bible lived approximately 900 years until after Noah's Flood, at which time the age dropped to 120 years. It has further dropped to our present day average of about 70 years since the time of King David of Israel. Some have conjectured that this rapid increase in ageing after Noah's Flood was the result of an increased rate of mutagenesis due to increased ultraviolet (UV) exposure.[111] While it is clear that UV exposure can result in cancer of the skin (people with the disease xeroderma pigmentosum are extremely sensitive to UV light as they lack an adequate DNA repair mechanism due to a mutation),[112] this is probably not the major cause. However, the point is that there is documented evidence from several sources that the lifespan of man has decreased over the last 4,000-6,000 years.

Another potential indicator of animals being more healthy and longer lived in the past is that more and more giant fossil forms of living groups are being discovered. These vary from giant reptiles to giant mammals, birds and plants.[113-114] We know even now that animals and man have the capability and can be manipulated to grow larger. For example, mice which had a human growth hormone inserted when they were at the fertilized egg stage develop to the size of rats.[115] Humans with certain hormone disorders can grow up to 8-10 feet tall.[116] However, it can be said that one common effect of many disease conditions and/or adverse environmental conditions is a general decrease in body size. Since body size has apparently decreased with time in many if not most living things, this could be a reflection of degeneration.

A good example of how this degenerative process can be speeded up is by artificially inbreeding animals. The result is called 'Inbreeding Depression'. It is the result of increasing the homozygosity of the gene pool, which thus increases the likelihood of the expression of harmful recessive traits. "Breeders have long known that inbreeding is disadvantageous, that is, it depresses performance of most traits and causes deterioration in vigor and health. Also, lethals and other undesirable traits appear more frequently under inbreeding. This is caused by the increased proportion of homozygous loci."[117] Brown has stated that, "...increasing the inbreeding coefficient by 10% produces a 5-10% decline in a particular reproductive trait. Considering total reproductive performance the decline in fitness jumps to a staggering 25% or so for species which have not been extensively inbred in the past."[118]

Someone might ask, "why inbreed animals or man then?" For several reasons:

— For certain commercially valuable traits (for example, milk production, etc.);

— For certain cosmetic traits (for example, the Bulldog's facial look); and

— For social reasons (for example, certain 'races' of man inbreed — Caucasian, Negroid, Jew, Italian, etc.).

In each of these situations inbreeding is known to have a deleterious effect.

For example, with extensive inbreeding the milk production of cattle or the egg production of chickens decreases.[119]

Concerning the cosmetic look of the Bulldog, Blogg and Allan have commented, "The Bulldog is only one casualty of man's capacity to breed dogs

according to his whims. Sometimes the aim was to produce a breed for a specific objective trait but usually it was merely to suit the fashions of the time. Frequently these fashionable whims have cost the dog dearly in other ways. Breeders, in their quest for less important qualities such as coat colour, have lost sight of an overall, good, healthy dog."[120] The Bulldog has problems with smelling things, his teeth are crowded and prone to gum disease, and the excessive folds in the flesh around his face make him more vulnerable to skin diseases.

Man in his desire to inbreed has brought upon himself an enhanced frequency of disorders among certain 'races'. Negroids have an increased incidence of Sickle Cell Anaemia (1:400) and Thalassaemia (8:1000). Caucasians are more prone to Cystic Fibrosis (1:2500). Some Jews are affected by Tays-Sachs (1:3600; non-Jews 1:36000). Among Italian/Americans or Greek/Americans the incidence of Thalassaemia Major is 1:400.[121]

Probably the best documented example of the effect of gene mutational load is that found when one parent who is heterozygous for Sickle Cell (that is, has the trait) conceives an offspring with the mate who is heterozygous for B-Thalassaemia.[122–123] The resultant offspring can have a condition as severe as the homozygous Sickle Cell Anaemia. Thus, while both parents are only mildly affectecd by their condition, the additive effect of the two mutant conditions can severely compound the problem.

It is interesting to note that malaria infested regions cover large portions of Africa, the Mediterranean, and South East Asia (Figure 21). Both the Sickle Cell Trait and the Thalassaemia Trait appear to offer the affected individuals some resistance to malaria. However, the frequency of these two conditions in malarial regions are by and large mutually exclusive. That is, where the B-Thalassaemia Trait is present in high incidence, the Sickle Cell Trait is not present or is present in very low amounts. This may be due to the compounding effects of gene mutational load.

Thus, gene mutational load is present in all populations of animals so far tested. This is shown by the effects of 'Inbreeding Suppression'. It is also likely that the gene mutational load is increasing with the passage of time. This has not been generally proven, but is a likely hypothesis since many mutations occur as recessive traits and they are thus extremely difficult to rid from a population.

If the majority (99%) of mutations are harmful, and the frequency and diversity of these is increasing in each animal and plant population, the overall effect would be one of deteriorating the overall vigour of the population. Natural Selection may impede this process, but it is still compatible with the Second Law of Thermodynamics. Evolution, however, requires that organisms have evolved from simple to complex, which is in direct contradiction to the observations of gene mutational load. The present observations would be compatible with the concept that the natural universe and living things specifically were more complex, more healthy in the past, and this is consistent with the creationist philosophy.

6. (a) Speciation

What is a species and why is it important to the Creation/Evolution question? E. Mayr has answered the latter question, "All the available evidence indicates that the origin of the higher categories is a

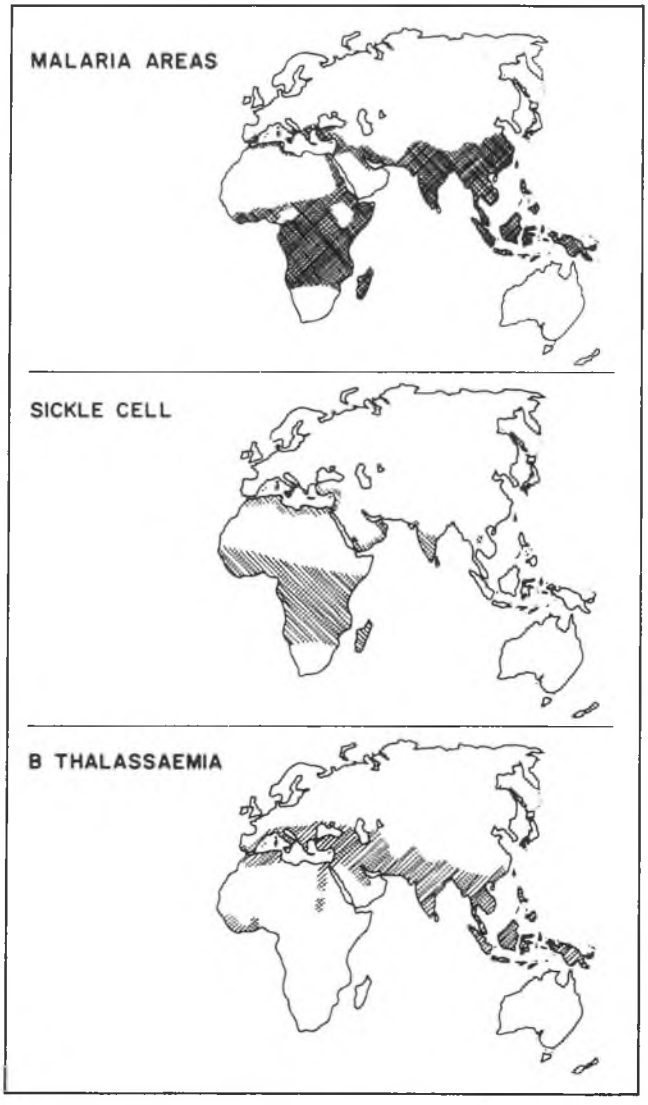

Figure 21. *An example of the effect of gene mutational load — B Thalassaemia/ Sickle Cell. Shaded areas indicate where the Sickle Cell trait or the B Thalassaemia trait is found. Malaria is present in both regions (cross-hatched areas).*

process which is nothing but an extrapolation of speciation. All the processes and phenomena of macro-evolution and the origin of the higher categories can be traced back to intraspecific variations, even though the first steps of such processes are usually very minute."[124] And mutations are the supposed source of any 'new' characteristics that will help differentiate species. Thus, the yardstick of evolutionary change is the so-called process of speciation. Gardner answered the former question of what is a species by defining a species as, groups of "interbreeding natural populations that are reproductively isolated from other such groups."[125] Thus speciation is the supposed process by which a common population of organisms are separated (reproductively or by physical isolation) such that each group develops distinct characteristics and with time will no longer interbreed with the other group(s) (Figure 22).

F. Ayala, I. Bock, and others have made extensive use of the concept of reproductive isolation to analyse the fly, *Drosophila*. Bock has noted that by mixing flies from different regions or habitats together:[126]

(1) "In many cases, of course the males and females concerned have simply refused to copulate"; or
(2) "Successful crosses have yielded results ranging from larvae that die before pupation, through pupae that fail to enclose, to production of adults, showing varying degrees of fertility and sex-ratio distortion."

Ayala divides these two groups of observations into Stage I (somewhat interfertile crosses) and Stage II (non-fertile crosses) speciation.[127] And from these observations 1500 species of *Drosophila* have been deduced (Figure 23). But the difficulty is, where does one draw the line between species that intermix reproductively and those that don't. This is difficult because the results are so variable.

Is this evolution in action? Not necessarily, for several reasons. The first is that a complete process of speciation has rarely if ever been observed in a natural population; and two, it is extremely difficult to define exactly what a species is. And thus, even if speciation occurs, the organisms still remain the same type, that is, *Drosophila*. It could even be argued as shown in Figure 22, that speciation is really a degenerative process that results not in the divergence of new life forms, but in the extinction of old ones.

Concerning the first, P. Parsons has commented, "In the sibling *Drosophila* species under consideration, there are possibilities for genetic analyses that may contribute to an understanding of speciation, but less than common are actual data.

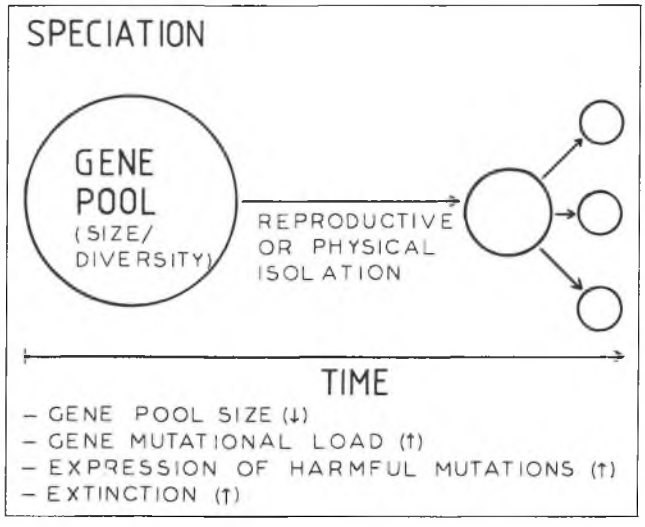

Figure 22. *Speciation. This process results in the separation of large (heterogeneous) gene pools into smaller more (homogeneous) ones. (↑) means an increase, (↓) means a decrease with the passage of time.*

Unfortunately, as Bush (1975) comments, 'The study of speciation is an ad hoc science, the reason being that no one has yet observed the development of a new species from beginning to end in nature'."[128] Thoday and Gibson, nearly 20 years earlier (1962), made a comparable comment, "Though speciation is one of the more striking features of evolution, direct experimental evidence concerning the origin of species is limited."[129] Again, it is interesting to note that Darwin, in 1863, said something very similar, "When we descend to details, we can prove that no one species has changed (ie, we cannot prove that a single species has changed); nor can we prove that the supposed changes are beneficial which is the groundwork of the theory."[100] This is a remarkable statement from the author of the 'Origin of Species'.

Thus, the process of speciation has largely **not** been empirically observed, but rather has been assumed to have occurred. *D. melanogaster* and *D. simulans* for example are naturally occurring populations. We do not know empirically whether or not they have a common ancestor (although this author is not necessarily saying that they do not). This is important when questions about the functional capability of the supposed common ancestor and the diverged species are desired to be compared.

It is also possible that *D. melanogaster* and *D. simulans* are not really different species but simply varieties within a 'kind'. The reason being the difficulty of defining a species. For example, in the *Drosophila* that do interbreed the results vary over a considerable range. When is one group of flies not of the same species as another? G.L. Stebbins, an evolutionist, has stated, "there do not exist in nature

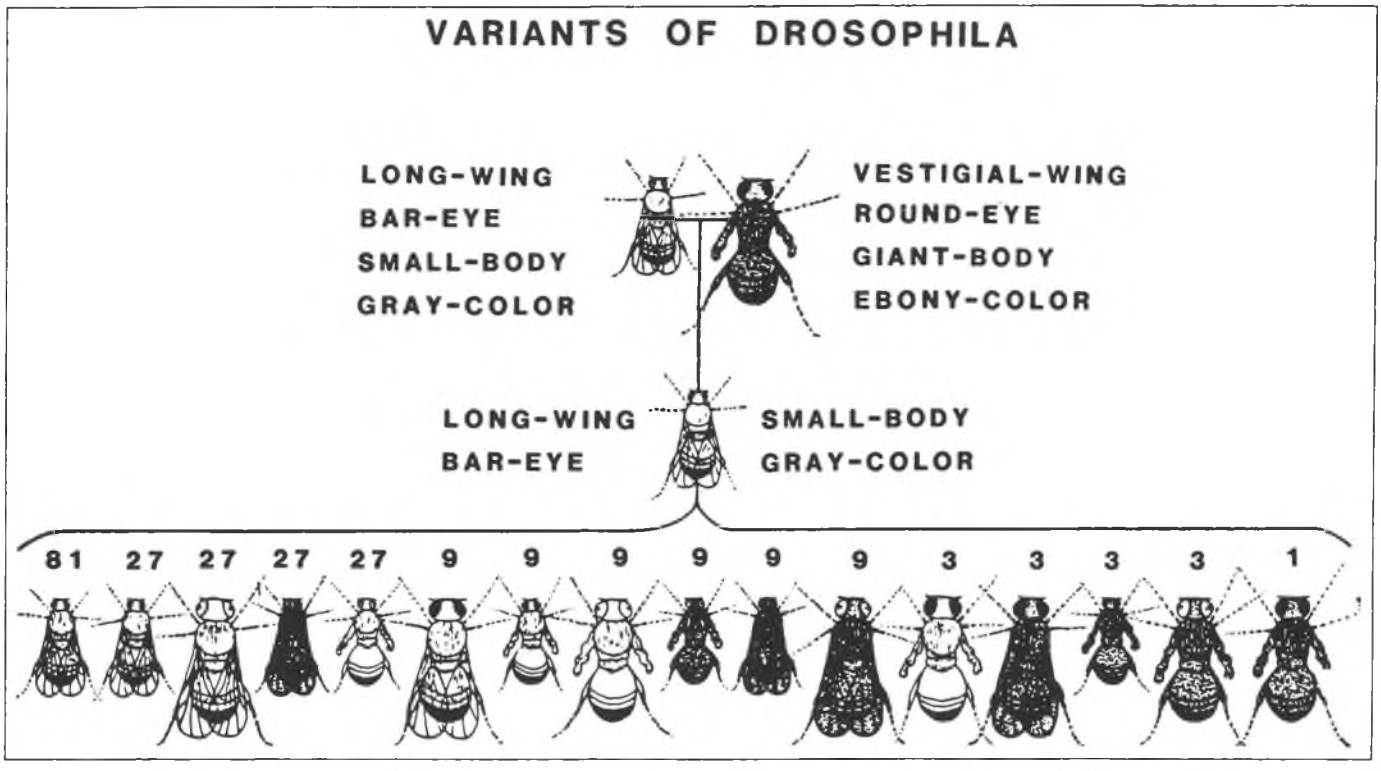

Figure 23. *The tremendous variety obtained in Drosophila* by the mixing of two flies with four different mutant and normal (wild type) phenotypic traits. The phenotypic results of the F_2 generation are:

81 long, bar, small, grey;
27 long, bar, small, ebony;
9 vestigial, round, small, grey;
9 long, round, small, ebony;
3 long, round, giant, grey;
27 long, round, small, grey;
27 vestigial, bar, small, grey;
9 vestigial, bar, giant, grey;
9 long, bar, giant, ebony;
3 vestigial, round, small, ebony;
1 vestigial, round, small, ebony;
27 long, bar, giant, grey;
9 long, round, giant, grey;
9 vestigial, bar, small, ebony;
3 vestigial, round, small, grey;
3 vestigial, bar, giant, ebony;

groups of individuals which must be grouped in only one way as objective, uncontestable species. On the other hand, species are not purely subjective groupings, carved out of an amorphous welter of varying populations. The organized systems of populations, forming an irregular variation pattern, is characterized by modes of similar variants separated by larger or smaller gaps of discontinuity. The best system for any group is one synthesized from data of all kinds."[130] Thus, reproductive capability is not the only criterion used by scientists for attempting to distinguish one species from another. In fact, *D. melanogaster* and *D. simulans* interbreed,[131] and "show a remarkable degree of morphological and genetic similarity."[132] Therefore, are they really two distinct species? P.Parsons thinks they are, but bases his conclusions more on their ecological behaviour, than on reproductive capability. He has stated that, "the two species are more readily distinguishable at the ecological level. . .The term ecological is here defined in the broadest sense to include behavioural factors involved in resources utilized and habitats selected in nature. Such biological divergences in the life histories of the two species must promote isolation between them into discrete habitats. . ."[132] Thus he suggests that microhabitat selection can be invoked as an important isolating mechanism. What he and others are advocating is not just a physical barrier, which Ayala might invoke for his Stage I of speciation. Rather Parsons is saying that within a certain group of organisms or plants there will be a sorting out process (behavioural) into various ecological niches. These would be called ecospecies which could but would not interbreed due to ecological barriers.

The observation that behavioural traits affect breeding patterns has also been noted by Tullar who has stated, "It is true that the members of one species do not interbreed under natural conditions

with those of another species except in rare instances. Under artificial conditions, however, it has been found that a number of species can be interbred with complete fertility, including various species of fish, the pheasant and the chicken, the dog and the wolf, and many others."[133]

If the concept of an ecospecies is acceptable then we would say that a Great Dane and a Dachshund are similar to ecospecies. It is known **within** written history that both come from the same stock.[134]

They are probably interfertile genetically but because of the size difference they are unlikely to interbreed physically. Thus, we could say that through artificial selection two **new** species have been obtained. So what, is this important? It is important because as Mayr has indicated, ". . .the origin of higher categories is a process which is nothing but an extrapolation of speciation." All the empirical evidence supposedly in support of evolution from the fields of population genetics, ecology, mutations, etc. rests on the concept of species and speciation. Did the Great Dane and Dachshund evolve into something new from an ancestral dog? Or are they just variants of these ancestors — dogs? This author would maintain that they are simply variants, as most probably are the 1500 so-called species of *Drosophila*. These variants may interbreed, or may not depending upon some of their physical and mental attributes, just as it is with dogs. This would allow for the development of mutations in some groups that are not common to other groups. But, just as artificial inbreeding increases the homozygosity of the gene pool and thus allows for the expression of harmful mutations, so true speciation would result in the same sorts of conditions — an overall decrease in the functional capability or adaptive potential of the organisms to future environmental changes.

Another example cited as evidence for speciation is that of the reproductive capability of leopard frogs from different parts of the U.S.A.[135] Frogs from adjacent habitats can interbreed, but frogs more distantly located, for example, New England and Florida, do not interbreed to produce viable offspring. Frogs more distantly located will probably be affected by different selection pressures and the gene mutational load in each may be different. Is this evolution in action? The answer is no. All the frogs are still leopard frogs. However, some can no longer interbreed, much to their own detriment, because the greater the heterozygosity of the gene pool (due to interbreeding), the greater the adaptability of those organisms. Thus, it could be argued that the leopard frog is in a process of fragmentation into less adaptable and therefore ultimately less viable variants (races).

As G.L. Stebbins indicated there are clearly different groups of living things, that is, cats, dogs, elephants, roses, apple trees, etc., that may have a number of variants within the group. But it is extremely difficult to categorize living things by just one criterion, that is, reproductive capability. (This may be because in the process of deterioration, certain variants may lose the flexibility to interbreed.) This difficulty is observed with regards to 'Darwin's Finches'. W.E. Lammerts has measured many specimens of the 100 different species and commented, "If one were to remove all the species labels and arrange the Darwin finches from largest to smallest in body and bill size, complete intergraduation would be found. The same is true of bill length and width. As mentioned above, there is complete intergraduation of plumage colouration although the smaller birds tend to have lighter grey feathers. The situation is exactly comparable to that of the song sparrow, *Melospiza melodia*, where one finds a comparable range in size of bird and bill. Here also the small desert forms are light grey in colour."[136] Therefore, are these finches really different species or just variants of one? Lammerts concludes, "If species are to be erected on such minute norms, then indeed we will be burdened with an almost infinite number of names. It seems much more in line with reality to consider these birds as all in one species, broken up into various island forms as a result of chance arrangement of their original variability potential, as regards the rather minor variation in bill and body size, skull features, and plumage colouration. A Sewel Wright random variation pattern would give exactly this sort of thing." The minute norms he is referring to is well shown in a recent publication comparing the shells of a living and fossil snail, *P. occidentalis* (fossil — Late Pliocene) with *P. cancellatum* (living), a difference of 10 million years (supposedly). The size comparisons were *P. occidentalis* 16.3 × 24.3 mm, 4-1/4 whorls (l/d 0.67), *P. cancellatum* 16.1 × 23.3 mm, 3-1/4 whorls (l/d 0.69).[137] Are these again just variants of the snail *Phortion*, or are they uniquely different species?

Frank Marsh has commented, "Our taxonomic experts list for us 64 species of blue grass and 160 species of panic grass in the United States; 17 species of common thistle and 51 species of violets; 24 species of willow; 54 species of oaks, and 153 species of hawthorn, 66 subspecies of deer mice, and 214 subspecies of the southern pocket gopher; 13 species of true cattle in the world, more than 30 races of the song sparrow in the United States; and 160 distinct breeds of man on the earth."[138] He feels that each of these aggregates of species could be variants within a 'basic type' and thus do not

represent evolutionary change at all.

The difficulty with classifying living things as one species or another is further exemplified by the act of S.J. Gould in reducing the list of 600 species in the genus *Cerion* into fewer than 20 'legitimate' species.[139]

It is also important to realize, within the present creation/evolution controversy, that species is not necessarily to be equated with the biblical kind. Harris *et al* have commented, "God created the basic forms of life called *mîn* which can be classified according to modern biologists and zoologists as sometimes species, sometimes genus, sometimes family or order. This gives no support to the classical evolutionist view which requires developments across kingdom, phyla, and classes."[140] Certainly the falcon and hawk producing after their kinds (Leviticus 11:14) indicates that a species is not synonymous with kind since each of these birds have further subdivisions. Reproductive compatibility is clearly related to the definition of a kind in biblical terms, but as has been shown this can be affected by a variety of factors (for example, artificial selection) which may be part of an overall degenerative process.

In conclusion, speciation is the supposed observable process of evolution, and mutations are the expected source of new raw material for speciation. However, there are few if any actual empirical observations on natural speciation. It may occur, but most reports on it simply assume it to have occurred, and assume the genetic inter-relatedness of similar but not identical organisms. It is also very difficult to define exactly what a species is. Excessive labelling of species is known to occur. And even if speciation occurs, what change is actually observed can be attributed to variation within a kind and not necessarily the 'minute' steps by which macroevolution (reptile to bird) is extrapolated. Finally, speciation can be viewed as a degenerative process that eventually leads to the extinction of organisms. Thus, to use speciation as the example of evolutionary change now occurring is unfounded, but it is compatible with the concept of an initial creation of distinct types of organisms with a tremendous potential for variability, and with variants able to interbreed more or less. It can also be argued that these distinct types of organisms within their variants are in a process of degeneration, and that this may hinder reproduction among some of these variants.

6. (b) Classification

"If we observe God's works, it becomes evident to everybody, that each living being is propagated from an egg and that every egg produces an offspring closely resembling the parent. Hence no new species are produced nowadays."[141] So said Carolus Linnaeus in 1735. Was he incorrect in stating this? As was noted earlier a species is very difficult to define. And while certain organisms may look very similar, they may have only limited reproductive interchange, for example, *Drosophila*. Some would note this lack of interaction as an indicator that the organisms were diverging into different types of animals (evolution), while the observations could just as easily be explained by stating that a process of deterioration has fragmented the organisms into less viable groups of the same organism (assuming that they had a common ancestor).

Other criteria besides reproduction have been used to estimate the 'relatedness' of organisms. Morphology and molecular homology (protein and DNA) being the major observational tools. Probably a better, more useful and less biased way to use these data would be in simply asking questions about structure/function similarities, rather than 'relatedness' in terms of who was whose ancestor. For example, the monkey has many design features similar to man. But to say that man actually evolved from a monkey is purely an assumption; it has never been and probably never will be observed.

However, some have assumed that all organisms are related by progeny, and that by mutations and natural selection the various forms of life diverged (or sometimes converged) through the vast periods of time. Thus they feel that it should be possible to compare differences in morphology and homology, and date when these changes occurred by using the fossil record. Then the rate of mutational change between various groups of animals and plants could be calculated.

It must be said emphatically that the above concept is purely a conjecture with no empirical basis. But even if we accepted it as a working philosophy, can the observations of science be harmonized with it? The answer is no, for several reasons which are numbered below (see also Figure 24):

(1) Does life come from a single line of descent or many? M. Dayhoff said in 1969, "The only reasonable explanation for the observed detailed biochemical similarities seems to be an evolution of all living organisms from a **single common ancestor** of all living things. . ."[142] (emphasis mine). However C. Schwabe in 1983 stated, "These and other discrepancies that can be found in the scientific literature **argue against a single line** of descent."[143] (emphasis mine) He advocates a cascade of life forms,

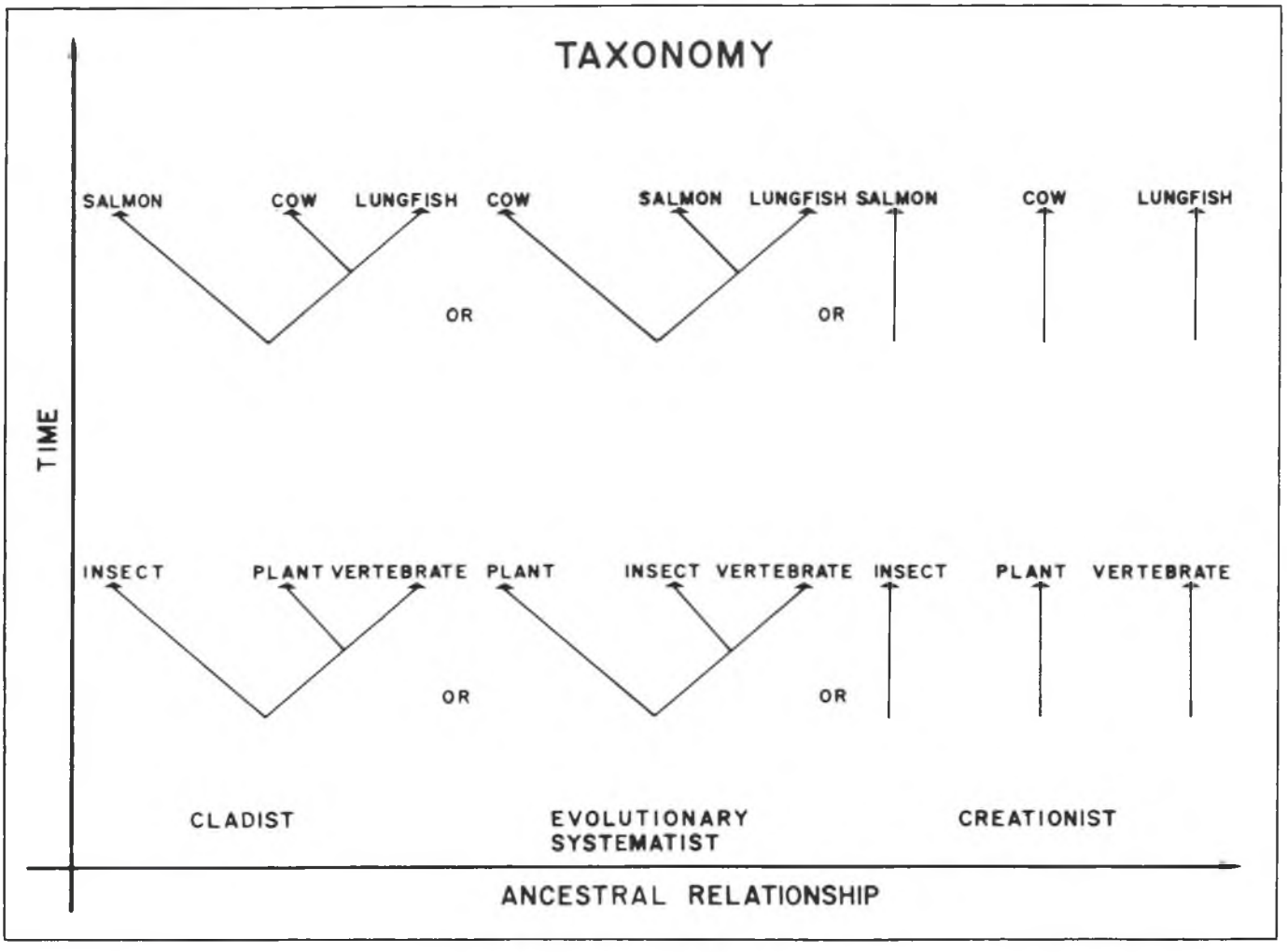

Figure 24. *Classification according to three current philosophies. Cladists use phylogenetic or phenotypic information. Evolutionary Systematists use a combination of both phylogenetic and phenotypic characteristics. Creationists use largely reproductive capacity, along with genotypic and phenotypic information.*

simultaneously evolving. If he is right then it becomes virtually impossible to trace any so-called ancestral relationships. But the more important point is that despite the massive amounts of data available on homology and morphology, these two leading scientists have come up with exactly opposite views. This discrepancy is **not** based on the lack of data as is the case in some controversies. The problem is with their assumption that all life forms are related by progeny. Since they can not empirically test relatedness, their opinions become pure conjecture. But secondly, it says that no easy straightforward, gradual changes are observed in either protein or DNA homology, or structural morphology.

(2) The above difficulty is reflected in the following statement: "The cytochrome c tree pictured in books and magazines is **only one of forty** trees generated by computer analysis of the data — the tree 'corrected' for closest fit to the 'known Phylogeny' (ie, the presumed evolutionary history). . .The computer must be told in advance to generate only ancestral sequences that allow for further ancestral sequences, otherwise, as we observed in some of our analyses, intermediate sequences are generated that break the presumed evolutionary chain."[144] (emphasis mine) In other words, the computer is told to make the data conform to the evolutionary order accepted by evolutionists.

There are many other discrepancies:

i. Alpha haemoglobin: chicken/crocodile 17.5% similar, viper/crocodile 5.6% similar;[145]

ii. Myoglobin: turtle/crocodile 11.8% different, chicken/crocodile 5.2% different, turtle/chicken 5.9% different;[146]

iii. Insulin: sperm whale/fin whale similar to those of dog/pig but differs from sei whale;[147]

iv. Cytochrome C: rattlesnake/turtle 21 per 100 amino acids different, rattlesnake/human 14 amino acids different; dog/screw worm fly 15 different.[147–148]

C. Patterson makes an accurate comment regarding the comparative study on myoglobin from turtle, crocodile, and the chicken. He said, "The theory (evolution) makes a prediction, we've tested it and the prediction is falsified precisely."[146] (insertion mine) For example,in evolutionary theory turtles and crocodiles should be more related to each other than to the chicken, but the myoglobin data indicates that each is more closely related to the chicken than to each other.

Also, using the cytochrome c data which animal is more related to the other, snakes and turtles or snakes and humans? To deal with this anomaly some make another non-testable assumption, for example, that some proteins within certain animals evolve faster that those within others.

As F. Leclercq has stated, "A difference thus appears between the phylogenetic tree based on fossils and that based on the number of substitutions in the haemoglobin primary structure. . .We are faced with two hypotheses: either the snakes separated from the crocodiles earlier than the mammals, or snake haemoglobin evolution has been quicker than mammalian."[149] He has no way of empirically testing either so they are not even hypotheses! But there is a third alternative — they were never related.

(3) The problem referred to in (2) has been further compounded by a presently propounded concept that in some cases of protein/DNA homology it is more important to use structure than primary sequence. Grütter has stated, "The ability to make meaningful comparisons between such distantly related proteins must therefore come primarily from structural homology, and only secondarily (if at all) from sequence homology. On the other hand, structural homology in the absence of sequence homology might be attributed to convergent rather than divergent evolution."[150] The implications of this concept are profound. No longer is homology necessarily based on the genetic coding of a protein, but now purely structural similarities of the protein can be assumed to be an indicator of relatedness. Thus, lysozymes from the Embden goose, chicken, and bacteriophage T4 which have little similar sequence, but have similar structure are considered to be related ancestrally. Also one can arbitrarily pick and choose whether you consider it convergence or divergence. Needless to say this sort of analysis could cause tremendous problems in classification.

(4) The results of DNA sequence homology present similar difficulties to those of amino acid sequence homology, except that they are more complex. Three base positions represent each amino acid, and there is some variability in the codons for some amino acids. C. Patterson has commented, "One last thing, at this level of DNA (mitochondrial DNA). . .we also have a problem of homology. What does homology mean in terms of DNA? The alignment procedure is the same with protein sequences, it's purely statistical business but because in DNA we only have 4 possible nucleotides in any one position, we expect a 25% match by chance alone. Among these 5 very closely related species (man, chimp, gorilla, orangutang and gibbon) there is only a 7% match, that leaves a 45% variation to accommodate all other eukaryotes. I think that the problems with aligning DNA. . .will be extremely (difficult). . ."[146] (insertions mine)

Another problem is that introns are present in genes from many organisms, but not in comparable genes from other organisms. For example, insect globin genes do not contain introns, vertebrates contain two, and plants contain three.[151] It could therefore be calculated from the observations on introns alone that plants and vertebrate globins are more related than vertebrate and insect globins (Figure 24). However, many would group insects as more related to vertebrates than plants when morphology or amino acid sequence homology criteria are used.

The conflict between DNA sequence and morphological studies is especially apparent in the currect attempts to assign the nearest relatives among the gorilla, orangutang, chimpanzees, gibbons and man. Sibley and Ahlquist have noted that five different "trees" have been proposed. They conclude, primarily from DNA studies, that man is most related to the chimpanzee and then the gorilla.[152] Schwartz on the other hand, uses morphological data to conclude that man is more nearly related to the orangutang, and he is not as closely related to the chimpanzee or gorilla.[153]

The conclusions from studies done on DNA sequence homology already indicate that it contains the same problems as protein or amino acid sequence homology studies, along with the further complications. For example, there are only four bases in the DNA code (which increases probability of match not due to relatedness), variability of codons, and presence

or absence of introns. A final point to be made is that the DNA sequence results often conflict with other sources of data (for example, morphology).

(5) Morphology has caused real problems for evolutionary classification. Living things that contain a mosaic of traits are particularly troublesome. For example, the lungfish has an air-breathing respiratory mechanism and a fish-like body. Should it be classified as more related to the cow or the salmon?[154] Some evolutionists (cladists) would associate it more closely with the cow, thus emphasising the morphological traits of respiration. Evolutionary systematists may, using other criteria such as the fish-like characteristics, classify the lungfish with the salmon (Figure 24). Who is correct? Or are the salmon, cow and lungfish simply not "related". Why not state that they are simply different, but reproduce after their own kind, recognizing that some organisms being debilitated by mutations and isolation may no longer interbreed with other 'normal' members of their group, but will still do so among the subgroup. Call this speciation if so desired, but let it remain simply variation within a type or kind.

In conclusion, it has been conjectured that the proteins, DNA, and morphology of living things can, when compared reveal the relatedness of present living things to supposed common ancestral forms and the rates of mutational change by which these organisms were supposed to have diverged. Again, it is purely an assumption, but even given that it might be true, when the analyses are made there are so many contradictions which require further secondary assumptions as to make the primary postulate falsified.

Maybe Linnaeus wasn't so wrong after all.

7. Fossil Record

The fossil record will only be discussed in relationship to the present study on mutations.

It has been stated by C.O. Dunbar that, "although the comparative study of living animals and plants may give very convincing circumstantial evidence, fossils provide the only historical documentary evidence that life has evolved from simpler to more complex forms."[155] Thus, the fossil record is the supposed direct evidence for evolutionary change. Or does it better support Creation? Evolutionists often neglect to note that transmutation from one organism to another is not empirically testable in the fossil record, but be that as it may, if the fossil record is consistent with evolutionary change then it should reflect the results of mutations causing this change.

In fact, it is known that mutations affecting bone structure are preserved in fossilized specimens.

For example, the human femur bone located at the site of the so-called Java Man contains an outgrowth probably due to a bone cancer.[156] One Neanderthal Man skeleton was probably affected by osteoarthritis or some other bone disease, which would have caused the bone deformities seen in that specimen.[157] Therefore, mutations affecting bone structure have been detected in fossils. It is true that many mutations would not be reflected in bone structure, but it is also clear that macroevolutionary changes from a reptile to a bird would require many major bone changes. It is also likely that these changes would have had to be small and would have had to be gradually accumulated, for as has been shown natural selection is very efficient in removing gross disorders. Therefore, there should be many transitional forms, incrementally changing from one form to another. And since this must occur among all living things, millions of gradual (incremental) transitional forms would be expected to be found in the fossil record. Creation, on the other hand, would postulate a stasis of organisms in the fossil record, with no transitional forms (except variations within the kind, of course). Mutations in bone formation would simply result in deformities to a living organism, and these organisms if fossilized would appear as deformed members of a known group of fossil organisms (for example, Neanderthal Man).

One observation that is immediately apparent is that there are gaps between virtually all the taxa of the fossil record (as is true for living forms). In Darwin's day it was thought that these gaps would eventually be filled in. However, since that time the gaps have become more sharply defined, and still remain. T.N. George has stated, "The fossil record nevertheless continues to be composed mainly of gaps."[158] Thus, Darwinism (gradualism) lacks evidence.

Therefore, a new philosophy (for it is untestable as a theory) has been expounded called Punctuated Equilibrium. It is postulated that a group of organisms remain static until some environmental (or other) force acts on them and a mutated form becomes predominant. The change is rapid and extensive so that few if any transitional forms become fossilized. This is Goldschmidt's hopeful monster or bird out of a reptile egg concept.[83] Again, this philosophy lacks empirical observation because it explains why there are no transitional forms, but how is it known whether there were any to begin with? What it also implies is that massive mutational changes occurring over a relatively short period of time can be helpful, and actually lead to increasing complexity. Needless to say this idea suffers more

problems than gradualism in that more massive, rapid changes are needed. The observational effects we have of mutations clearly indicate that large mutational changes are generally more severely debilitating. For example, in cattle mutant 'bulldog' calves are severely deformed, whereas mutations affecting coat colour are not so nearly debilitating. It would strain one's imagination to suppose that more massive mutations could and even were necessary for evolution. And even if the change occurred through a series of 'smaller' mutations over a short period of time as some may postulate, the problem of the probability of successive helpful mutations and gene load argue against this. Punctuated Equilibrium only aggravates the problem of evolutionary change by mutations.

A second pertinent observation is that there are so-called 'living fossils' among many taxa of animals and plants (Table 3). S. Stanley has stated, "The remarkable geologic longevity of species has only recently come to light. . .for fossil faunas of marine bivalve mollusks 7 Myr (million years) old, approximately half of the species are so similar to living forms as to be judged conspecific. . .approximate ages of fossil faunas comprising 50% extant species are as follows: marine gastropods, 3.5 Myr; benthic foraminifera › 15 Myr; planktonic foraminifera › 10 Myr; freshwater fish, terrestrial mammals, 0.7 Myr. For beetles (taxonomy based on faithfully preserved genitalia) nearly all species younger than 2 Myr are extant. . .approximate ages of floras in which 50% of the species are extant are: seed-bearing vascular plants (taxonomy based on seeds), 4 Myr; marine diatoms, 12 Myr; and bryophytes › 10 Myr (nearly all known Miocene and Pliocene species are extant)."[159] Probably the classic example is the Coelacanth which has not changed for presumably 300 Myr. This is highly unexpected given the present observations on mutations for several reasons:

(a) Mutations appear to be affecting all plant and animal life. This effect appears to be additive and deliterious (gene load), and it can be measured in terms of a few life-spans of the living thing. It is simply unlikely that Coelacanths (among many others) would remain uneffected morphologically for 300 Myr or approximately 6 million life-spans (if 50 years equals one life-span). Yet Hickman blithely passes this off saying, "A striking similarity between living Latimeria and fossil coelacanths of 300 million years ago indicate that these fish have been able to adapt to a changing environment without structural changes."[160] The observations of science argue directly against his statement. If however, the fossil fish was not nearly so old and

Table 3: Animals and Plants that have not changed morphologically since their 'supposed' appearance in the geologic record

NAME (Common)	*NAME (Technical)*	*SUPPOSED TIME SINCE FIRST APPEARANCE (MILLION YRS:MY)*
Mammal		
Bat	*Icaronycyeris*	50
Pangolin		35
Tapir		20
Bird		
'Modern Bird Fossil'		› 150
Reptile		
Lizard	*Tuatara*	135
	Lanthanotus borneansis	MY
Soft-Shelled Turtle		60
Alligator		35
Amphibian		
Frog		› 70
Siren		70
Fish		
	Coelacanth	90-400
Port Jackson Shark		180
Cow Shark		166
Cat Shark		135
Bowfin		65
Mollusca		
Clam	*Neopilina galathea*	500
Squid	*Spirula*	200
	Vampyroteuthis Infernalis	100
Nautilus	*scrobiculatus*	570
Snail	*Trigonephrus globulus*	10
Echinodermata		
Starfish		› 50
Sea Lily	*Rhizocrinus lofotensis*	160
Arthropods		
Spider		MY
Myriopod — Centipede/ Millipede		› 100
Mite		70
Arthropod/Crustacean		
Horseshoe Crab		225
Insect		
Cockroach		120
Ant		120
Fly		70
Japanese Cupes Beetle		120
Bacteria		
	Archeobacteria	› 100
Ctenophore		MY
Algae		
Stromatolites		› 500
Trees		
	Meta Sequoia	
	Glyptostroboides	60
	Ginkgo	200
	Cycad	200
Hickory		10
Japanese Oak		55
Walnut		120
Magnolia		120
Vines		
Grape		120
Various Plants club mosses, horsetails, ferns, liverworts, mosses, hornworts		400

From:
(1) R.L. Wysong — Ref. 103.
(2) Science Digest (1982), p. 92.
(3) A.C. van Bruggen — Ref. 137 (snail — **Trigonephrus globulus**).

if the 'kind' never changes (as creationists propose) then the similarity between it and the living fish might be expected.

This similarity which might be called stasis (that is, no change) is characteristic of all major groups of life. Professor Carter has commented, "generally each of the major life groups has retained its fundamental structural and physiological characteristics throughout its life history and has been conservative in habitat."[161] This would **not** be expected if each of the groups were millions to hundreds of millions of years old. More variant (mutated) forms as well as transitional forms would be expected.

(b) The fossil record is probably not as old as many evolutionists have proposed. H. Morris has listed seventy different ways that the age of the earth has been estimated — ages vary from zero to five hundred million years.[162] Others have estimated an age up to 3.9 billion years.[163] However, Whitcomb puts forth a strong case as to why the earth should be estimated to be around 10,000 years old.[164] In any case, the age estimations vary dramatically and they all rely upon non-empirically deduced assumptions. Thus, it could be said that all of the dating methods are subjective, and probably the main reason long age estimates are popular is that it would require vast amounts of time for major changes to occur by mutations. However, since the effects of mutations are largely degenerative, and appear to increase with time, long periods of time would only aggravate the problem. Thus, it is more likely from these sorts of observations that the earth has a young age.

(c) A final consideration is that of the size of fossil plants and animals. It would be expected that if life arose from simple organic compounds to single-celled and then multicellular organisms that there would be a corresponding increase in both complexity and size. In fact, the increase in brain capacity from opossum to man is used by some as an indicator of evolutionary advancement.[165] However, the fossil record reveals many contradictions to this concept. For example, there are giant fossil forms of many animals,[113–114] plants and possibly man.[166] Thus, the living things of the past may have been larger, longer-lived, and more abundant. It is interesting to note that the Bible as well as secular sources comment on giant men who once roamed the earth.[167] And while it is clear that mutations can cause both gigantism and dwarfism through metabolic disorders, the observation of such a widespread diversity of ancient giant fossil forms is inconsistent with the generally accepted evolutionary advancement of living things. But it is consistent with a degeneration of life forms from long-lived large bodies to shorter-lived smaller bodies (albeit this is still largely conjecture).

In conclusion, the fossil record of stasis in life groups and 'living fossils' do not support the concept of evolutionary development by mutations from simple to complex over hundreds of millions of years. It supports better the concept that the age of the earth is much shorter, and thus mutations along with gene load have not yet caused extensive degeneration in life forms.

D. A GENERAL CONCLUSION

Concerning origins, science cannot directly give a categorical answer. But present observations may be used to postulate what might have happened in the past.

One point that the study of mutations clearly reveals is that all life is in a process of deterioration. The observations relating to the general effects of mutations, design/optimized structures, probability, natural and other kinds of selection, gene load, speciation/classification, and the fossil record all concur with this. This is compatible not only with the Second Law of Thermodynamics but also with common sense!

Thus the empirical observations on the effects of mutations are one of the strongest testimonies that refute evolution philosophy. But they are compatible with a corollary of the creation philosophy which states that after life-forms were initially created as complex, individual, reproducing organisms, deterioration began and continues to the present. The Bible says that in the beginning what God created was good, but that through one man's sin (Adam's) death entered the earth and spread to all men (Genesis 1 and Romans 5:12). That this sin affected all of the physical creation is evident for Romans 8:20–22 says, "For the creation was subjected to futility, not of its own will, but because of Him who subjected it, in hope that the creation itself also will be set free from its slavery to corruption into the freedom of the glory of the children of God." Therein lies a great hope, the redemption of man and nature by God through Jesus Christ.

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